CLINICAL ELECTROCARDIOGRAPHY

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By

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PREFACE

A SERIES of excellent books on electrocardiography is already in existence and the responsibility in adding to the voluminous literature on this subject is great Therefore the authors consented to undertake this work only after considerable hesitation

A knowledge of electrocardiography presupposes an acquaintance with a large number of rules and laws The acquisition of these facts requires the expenditure of considerable time and effort but otherwise is not difficult These general facts have been adequately presented in most text books on electrocardiography Owing to the novelty of the subject and the necessity for presenting the fundamental features of electrocardiography to readers unacquainted with this field writers have very properly limited themselves to essentials Consequently an unwarranted notion of the extreme simplicity of electrocardiography has developed among those whose contact with tracings is infrequent For this reason it seems timely to present not only the usual rules but also to emphasize some of the difficulties encountered in endeavours to apply them Since these factors are introduced this book is not designed primarily for those beginning their study of this field Moreover a departure has been made from similar works by the introduction of considerable clinical and therapeutic material It is hoped that this device will create a closer alliance between the electrocardiography and the clinic While the limitations of the electrocardiogram find emphasis this alliance may extend the usefulness of this method In several fields of electrocardiography the widespread acceptance of some theories has unintentionally led to the notion that these useful working hypotheses have been established as true At times we have ventured to present incompletely solved aspects of these situations with the hope of stimulating further investigation

Fortunately the cost of the apparatus is no longer prohibitive and the registration of the electrocardiogram has become greatly simplified This is reflected in the rapid increase in the number of physicians who employ the electrocardiograph This situation is not entirely devoid of danger Considerable practice and patience is necessary before one can recognize the alterations at times very slight in the design of the electrocardiogram as something definitely

pathological and can distinguish them from the normal picture with its innumerable variations Time and patience are not at the disposal of everyone Not rarely the interpretation is reported without the necessary knowledge, often the individual findings are regarded dogmatically and the heart is stated to be healthy or affected with great positiveness The difficulties or uncertainties associated with electrocardiographic interpretations often are not adequately stressed If the physician who receives such reports is unacquainted with these limitations, he may be induced to institute or fail to institute measures which may vitally affect the fate of the patient

This circumstance alone constitutes an important reason for physicians who are not particularly concerned with electrocardiography to know its basic rules, so that they may evaluate the findings reported to them In this connection it should not be forgotten that despite the study of countless tracings by innumerable observers, many phases of electrocardiography are still in the formative phase The exact criteria in borderline cases, for example, the height and width of individual waves, remain unsolved and important problems

For a long time students of electrocardiography were almost exclusively occupied with the arrhythmias which were, for the most part known prior to the introduction of electrocardiography In recent years so many new and valuable advances have occurred in the electrocardiographic study of myocardial diseases that the electrocardiograph has become indispensable in the examination of these common problems The present knowledge of the alarming incidence of myocarditis, the early diagnosis of the coronary artery disease, the possibility of recognizing atypical cases of coronary thrombosis would have been impossible without electrocardiography Physiology and anatomy as well as the clinic had been enriched by numerous advances of fundamental importance

Although the electrocardiography is frequently and widely employed, it deserves more extensive use It *alone* may permit the physician to recognize the participation of the heart in diabetes, diphtheria, tonsillitis and in many other conditions The electrocardiogram alone may reveal the existence of coronary artery disease in patients who complain simply of abdominal fullness and flatulence in the absence of "cardiac" symptoms The discovery of electrocardiographic changes after exercise, indicative of profound disturbance of myocardial blood supply, and necessitating the diagnosis of extreme coronary stenosis in patients with vague substernal distress and clinical signs of a "minimal aortitis" is, we believe,

another important advance suggestive of the great possibilities of electrocardiography.

Electrocardiography is still a young science. It was discovered and developed within the memory of many now living. Every year witnesses an abundance of new discoveries. Nevertheless many fundamental problems are still unsolved; others which seemed satisfactorily clarified in the recent past have become dubious in the light of new investigations which have revealed unsuspected complexity and the necessity for further study. An excellent example of this situation is provided by bundle branch block which seemed to be a closed and minor issue. The excellent work recently performed in this field has opened very intriguing vistas. This situation conspires to make comprehensive presentation obsolete at an early age. It also hampers authors since contrasting opinions on important and unsettled problems render their task difficult and tend to confuse the reader.

For this reason it has seemed best to omit some important and interesting questions, such as the problem of excitation, the connection between contraction and the electrocardiogram. On the other hand, some stimulation may be derived from very brief discussions of the development of extrasystoles, of fibrillation, and of conduction disturbances. Those who wish to delve more deeply into these problems will find suggestive bibliographies appended to various sections of the text. These references do not pretend to be exhaustive; in general, publications reflecting the present status of our knowledge and containing additional bibliographies have been given preference.

Emphasis should be placed upon one important rule of electrocardiography which is frequently violated, namely, great care should be exercised in drawing conclusions from the electrocardiogram alone, without knowledge of the patient. Not every inverted T wave in Lead I has the same meaning! This finding cannot be evaluated until the interpreter knows whether or not the patient has received digitalis, whether hypertension is present or absent, etc.

It is also improper to make *anatomical* diagnoses from the electrocardiogram. Under ordinary circumstances the electrocardiogram assists in establishing the presence of foci in some area of the myocardium; it does not inform the observer concerning the nature of these foci. There are no typical coronary waves, no electrocardiograms typical of coronary thrombosis, of hypertrophy, of coronary insufficiency, of anoxia, etc.

The recent discovery that the T waves may become altered by

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diseases of other organs (for example, of the lungs, gall-bladder), since vago-vagal reflexes influence the blood supply of the heart, and the fact that several minutes of quiet standing by some untrained individuals can lead to a disturbance of cardiac blood supply and to abnormal electrocardiograms, opens new possibilities. It is possible that, at times, the severe electrocardiographic alterations or the heart failure in pneumonia, the rapid death after aspiration of foreign bodies will be traced to the participation of similar reflexes.

The problem of nomenclature also presents considerable difficulties, since entirely different names are employed in many places. An international agreement is urgently necessary.

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GENERAL REMARKS ON THE ELECTROCARDIOGRAM

THE ELECTROGRAM AND THE ELECTROCARDIOGRAM

If a strip of muscle is stimulated at its end A by means of a shock from an induction apparatus, the muscle is "excited" or activated *at this point* (Fig 1a) *The excitation is then rapidly transmitted over the entire strip towards* B

Electrical forces are awakened by the excitation of living tissue According to the classical theory of physiology every excited portion of the muscle becomes electronegative in relation to its unexcited part just as in a battery the zinc electrode becomes electronegative to copper If the two ends of a conducting wire are applied to the ends A and B of a muscle strip (Fig 1a) at the

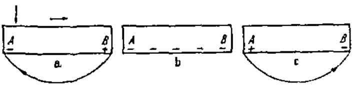

Fig 1a c Diagram of the spread of excitation in a strip of heart muscle

moment A is excited a current flows in the wire from B to A from the site of higher to lower potential If a galvanometer is included in the circuit it will register a definite deflection in a definite direction When this deflection is registered graphically, a wave is obtained which is directed upward (positive) providing the connections to the galvanometer have been made with the correct polarity (Fig 2)

If the excitation has affected the entire strip between A and B they are equally excited At this time the same potentials exist at both ends (Fig 1b), no current *flows through the galvanometer*, the apparatus records no deviation, and a zero (O) *or isoelectric* line is recorded

Recovery from the excitation begins very soon Naturally the

travels over the heart at the same time as the excitation Thus, it was found that in excited tissues potentials also appear which are *positive* in comparison with unexcited tissues (Lewis, Craib) Because of these results it was assumed that the excitation causes the development of a "double potential" in which the positive and negative poles lay close to *each other (at the junction of the excited and unexcited tissue or even* in the same muscle element) However, objections have also been raised *against this interpretation and at all events the finer mechanism is still* obscure

THE ELECTROCARDIOGRAPH

Principle of the Apparatus

If the thorax and pericardium of a dog are opened and the nerve of a nerve muscle preparation is applied to the surface of the exposed heart, the muscle innervated by the nerve contracts with the same rate and rhythm as the heart (Kölliker and Muller, 1856) In this simple and excellent way it was proven that bioelectric currents are produced by cardiac action

The following observation is *another example of the same fact* during an animal experiment, contractions of the left half of the *diaphragm are often noted which have exactly the same rhythm as* the heart They vanish when the left phrenic nerve is lifted or cut below the point at which it touches the heart This observation also proves the development of electrical potentials by cardiac action

Waller studied and also measured those potentials which reach the surface of the body Owing to its inertia the apparatus (capillary electrometer) employed by him was inadequate, and accurate records were only obtained when Einthoven invented the string galvanometer

The *principle of the apparatus is simple and generally known* if a magnetic needle is suspended in the vicinity of a circuit through which a current is flowing, the needle is deflected, the amplitude and direction depending on the direction and strength of the current If a powerful fixed magnet is employed and a fine movable conductor is brought into its field, the conductor will be deflected when a current flows through it The second principle is employed in the string galvanometer A thin metal thread or a quartz fibre coated with metal (the string) is suspended between the two poles of a *powerful electromagnet.* "*The action current of the heart*" led from the person under examination is conducted through this string, whereupon its movements correspond to the direction and strength of the current These movements are magnified many

excitation fades away earlier in the muscle end A which was stimulated first for this reason B now becomes more negative than A a current flows through the apparatus in the opposite direction (Fig 1c) and a downward directed (inverted or negative) wave will be recorded (Fig 2)

Accordingly a diphasic tracing can be obtained from a muscle strip of suitable length and breadth with properly arranged leads in it one wave is directed upward and the other downward and the two waves are separated from each other by a short isoelectric line Such a tracing obtained by *direct* leads from an excited muscle is called an *electrogram* or Eg

Knowledge of this simple electrogram of a muscle strip facilitates the understanding of the more complicated currents of cardiac action obtained in man The first major difference consists in the fact that the human heart is composed of a very large number of muscle bundles varying in size running in most diverse directions and interweaving with one another in manifold ways A second and very important difference lies in the type of derivation In clinical examinations the electrodes cannot be applied directly to the heart in order to record the tracing

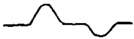

FIG 2 Electrogram from a strip of heart muscle

During the spread of the excitation wave over the human heart considerable differences in potential appear For the most part they are neutralized by the blood inside the heart and by the surrounding tissues Only a fraction of the potentials reaches the surface of the body where they can be intercepted and measured This type of lead is *indirect* and in contrast to the electrogram the tracing is called an *electrocardiogram*

While the amplitude of the deflection in the electrogram depends somewhat upon the width and thickness of the muscle strip and is proportional to it this relationship is absent in the electro cardiogram In the latter only that fraction of the potentials formed by the heart which can reach the body surface are recorded

In the electrocardiogram the position of the heart in relation to the sites of derivation and the status of the tissues surrounding the heart exert a very marked influence on the potentials which reach the surface of the body These influences have been studied in detail by numerous model experiments (Lewis Craib Schellong and others)

On the basis of experiments on simple muscle strips in recent years doubt has been expressed in regard to the theory that a wave of negativity

Rest Current, Polarization

Owing to the activity of the skin glands and through alterations in the innervation of the cutaneous vessels disturbing differences in the potential appear between the electrodes and give rise to the development of rest currents Without the use of compensating counter measures these constantly displace the string from its zero position and may make the registration of an electrocardiogram impossible

Two methods are used to counteract the disturbance caused by rest currents In some apparatus a *condenser* is connected with the galvanometer the former absorbing the very sluggish and slowly varying rest currents This method has the advantage of facilitating rapid work but possesses the disadvantage that it may considerably deform the tracings The sluggish waves (T waves) are most affected (see p 24) For this reason a second method is commonly employed *the rest current is compensated by means of a device capable of sending a counter current of equal strength through the galvanometer This method may necessitate the* employment of a switch during the registration of the electro *cardiogram but has the advantage of securing undeformed records* With the amplifying type of apparatus compensators are unnecessary

Unsuitable electrodes may by polarization give rise to serious distortion and by changing the shape of the initial and terminal deflections suggest a pathological electrocardiogram in an healthy individual For this reason with the string galvanometer one must strictly follow the directions accompanying the apparatus in regard to the type of the electrodes and the manner in which they are applied Electrodes made of German silver and of definite size (not less than 15 sq cm) are most suitable The skin should be thoroughly rubbed with alcohol cloths which have been immersed in a warm saturated solution of sodium chloride are placed between the electrode and the skin Recently an electrode jelly has been introduced for the same purpose and has proven to be very useful

Time Recording

In order to measure the single events registered in the electro cardiogram or to determine the cardiac rate a time record is photographed on the film simultaneously with the electrocardio gram This also is necessary because the film in the camera may not always move at the same speed Differing types of apparatus

times by a microscope and are projected into a camera where they are photographed on a film or strip of paper A small slit with a lens in the camera aperture makes it possible to project the movement of a small section of the string on the sensitive paper

Formerly the apparatus was quite cumbersome and difficult to transport since the magnet required considerable space and had to be maintained by heavy storage batteries This was necessary in order to obtain the requisite sensitivity In recording the electro cardiogram potentials are involved which rarely exceed 2 millivolts More recently the amplifying tube principle employed in the radio industry has also been applied to electrocardiography and electro cardiographs capable of amplifying about 3 000 times the feeble cardiac currents reaching the surface of the body have been constructed They weigh but little and can be transported easily (see p 25)

The description of the individual types of apparatus can be omitted there are several useful forms and any physician can obtain all the details by securing a prospectus from the manu facturers The principle is always the same and the operation is easy Portable electrocardiographs of both types (string and amplifying) are now available

Standardization of the Apparatus

Before the registration of an electrocardiogram the apparatus must be standardized so that comparable tracings are obtained If string galvanometers are used the patient should be included in the circuit during standardization The resistance of the patient s circuit may vary due to changes in the moisture content of the skin and of the applied electrodes and this may require adjustment of the tension of the string

A known potential from a standard source is employed in standardizing the galvanometer This calibration is *usually* performed by recording the electrocardiogram in such a manner that a difference in potential of 1 millivolt produces a deflection of 1 cm on the film (normal sensitivity) For certain purposes greater sensitivi ties can be employed by decreasing the string tension in the string galvanometer or by greater amplification in the amplifying electro cardiograph Standardization is especially important in string galvanometers because marked slackening of the string seriously deforms the tracing

cardiogram with distinct deflections can be registered Following the counsel of Einthoven it is as a rule sufficient to lead from three regions of the body surface to obtain a complete series of electro cardiograms These points are the right arm, the left arm and the left leg

The electrocardiogram from the right to the left arm is called Lead I, or the transverse lead that from the right arm to the left leg which is approximately parallel to the cardiac axis is called Lead II or the axial (oblique) lead and that from the left arm to the left leg is designated as Lead III or the longitudinal lead The electrodes are connected with the apparatus so that the chief waves in the leads enumerated are *normally directed upward (positive)* The extremity which lies nearest to the base of the heart is called basal that nearest to the apex apical Leads are taken from the basal to the apical extremity that is from the right arm to the left arm (I) right arm to left leg (II) and from the left arm to the left leg (III)

In order to avoid confusion and to ensure that each electrode is properly placed they are usually distinguished by colours or markings To avoid the disturbing influence of polarization they must be of special construction and properly applied In addition to the three standard leads which encircle the heart only in the frontal plane additional leads are employed for definite purposes and will be considered later (see p 150)

THE DEVELOPMENT OF THE WAVES OF THE NORMAL ELECTROCARDIOGRAM

(Remarks on the Anatomy and Physiology of the Specific Tissue)

It is now proposed to describe the phenomena appearing in the electrocardiogram with the normal spread of excitation In describing the anatomy of the specific tissue only those features are considered the knowledge of which is indispensable for the understanding of electrocardiography

A distinction is made between two types of muscle fibres in the heart (1) the common (expulsion) muscle of the heart, (2) the specific or neuromuscular tissue

The specific fibres differ from the common or working muscle by virtue of a special property they possess the capacity to form stimuli in themselves alone and without external influence This is called *automatism* When placed under suitable conditions every specific fibre possesses this capacity (see p 165)

may employ a different time recording system so that the time intervals recorded in a particular case must always be stated The methods employed are to record the vibrations of a tuning fork the regular movement of a pendulum or the regular rotation of a spoked wheel controlled by a tuning fork, in the latter the spokes of the wheel are arranged at definite equal intervals so that the passage of the spokes before the lens casts shadows which appear as thin lines (ordinates) in the tracing The time base most widely employed is arranged so that the distances between two ordinates or between two tuning fork notches measure 0 04 second But there are also time markers so constructed that the distances between the two ordinates amount to 0 02 or 0 05 second, etc, thus whenever a tracing recorded upon an unknown apparatus is to be interpreted, the time base of the record must be ascertained

If the rate of a heart with regular rhythm is to be determined from an electrocardiogram the distance is measured between two ventricular beats or the duration of one cardiac cycle" and is determined in hundredths of a second To obtain the number of beats per minute the duration of one cycle in hundredths of a second is divided into 6 000 i e 1 minute expressed in hundredths of 1 second If the heart is irregular a number of cycles are measured and the average duration of a cycle is determined, 6 000 is then divided by the average value

With some time markers which are widely employed, the fifth spoke of the rotating wheel is thicker so that every fifth vertical line or ordinate is broader (Fig 35) The distance between two broad white lines is therefore 5 × 0 04 second, that is, 0 20 second A simple method is commonly employed to determine rapidly the cardiac rate in these tracings, the number of cardiac cycles between thirty thick vertical lines (6 seconds) is counted and this number is multiplied by 10 If 9½ cycles occur during this interval the cardiac rate is about 95 per minute For more exact measurement the method first mentioned is advocated

The Leads of the Electrocardiogram

Even the first investigators who led the current from the human body to a galvanometer found that deflections equal in size and shape were not obtained from all parts of the body If connections are made at two places located near to one another or which lie in a position symmetrical in a definite way to the cardiac axis, no deflections are obtained because the two areas are at the same potential There are innumerable places from which an electro

itself, rather the excitation spreads in all directions There are preferred connecting paths (simply because they are the shortest) to the left auricle, *e g*, the interauricular bundle (Bachmann), or below to the atrioventricular system One of them passes over the Torus Loweri (Fig 3) But in addition to this, numerous other pathways are present

Apart from the sinus node and its junctional fibres no specific tissue has been demonstrated in the auricle Collections of specific fibres are periodically described but it happens that groups of cells are found in several places in the auricle which histologically resemble

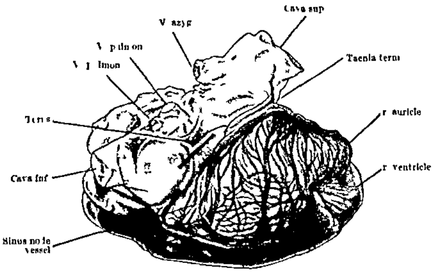

FIG 3 The base of a dog's heart made translucent by xylol viewed from the right (after Rothberger and Scherf) The site of the Torus Loweri is indicated by a dark line The Torus is connected with lower end of the sinus node A vessel embraces the sinus node in this instance

the cells of the sinus node Nevertheless experimental medicine has never been able to prove the existence of specific tissue in the auricle outside of the sinus node and its ramifications and the auricular part of the auriculo ventricular (A–V) node which is described below

As long as the excitation formed in the head of the sinus node remains in the node itself, no deflection from the isoelectric line occurs in the electrocardiogram The sinus node itself actually represents a very considerable collection of cells Nevertheless it is not sufficiently large to produce potentials which can be led from the surface of the body If in animal experiments punctate

There are two collections of specific fibres in the heart The first is the sino auricular node which in lower animals connects the sinus with the auricle and in higher types has become incorporated into the auricle The second is the atrioventricular system (see p 10)

The *sino-auricular system* consists of the sinus node (node of Keith Flack 1907) and its junctional fibres The broad upper end of the sinus node begins in the angle between the superior vena cava and the right auricle the lower tapering segment of the node extends to the region of the mouth of the inferior vena cava near the auriculo ventricular junction Its position is readily determined by means of these features In the opened auricle the sinus node is found even more easily In this instance one can easily distinguish a part of the auricle covered by trabeculæ from a smooth portion which corresponds to the sinus of lower animals The border between the two parts of the auricle is formed by a thick bundle of muscle the tænia terminalis The sinus node is found in the tænia terminalis

Fig 3 shows a drawing of a dog s heart after the preparation had been made translucent by xylol The division of the right auricle into two sections can readily be recognized to the left the smooth portion of the auricle may be seen to the right the remarkable network of trabeculæ In this heart the sinus node which begins in the auriculo caval angle is embraced by two vascular branches which arise from a vessel running downward to the right and anteriorly

In adult man the sinus node is 2 5–3 0 cm in length and approximately 2 mm in width it can be demonstrated macroscopically and is composed of the so called specific fibres which can be differentiated histologically from the common musculature by the fact that they are smaller contain more nuclei and are surrounded by much more connective tissue

Normally the cardiac stimulus arises in the head of the sinus node (pacemaker) Any other of the numerous fibres in the sinus node can also form stimuli however under normal circumstances all other fibres are silent only *one* of the many fibres (centre) lying in the head of the sinus node paces the heart The fact that *one* fibre among thousands suffices exemplifies one of the numerous safety devices in the heart

The connecting (junctional) fibres radiate out in all directions from the sinus node and soon imperceptibly merge with the auricular muscle There are no definite conduction paths in the auricle

of an entangled net of specific fibres which interweave in all directions.

Very often the connection of the A–V node with the auricular musculature is presented as if the node, lying in the right auricle, were alone connected with it. Actually numerous connecting fibres also run over to the left auricle. If an A–V rhythm exists in a dog experiment, then the left auricle contracts before the right (Rothberger and Scherf).

Gradually the cells become more parallel in arrangement, the structure of the specific tissue becomes more clearly defined, and

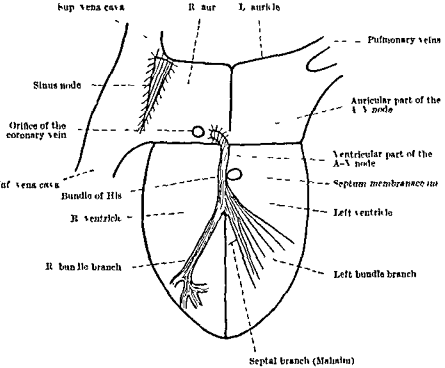

FIG 4 Schematic drawing of the conduction system

the A–V node passes into that section of the A–V system which is called the bundle of His. The bundle of His is easy to locate in the heart. It lies directly behind that part of the ventricular septum which contains only connective tissue and, for this reason, is transparent when held against the light (septum membranaceum, Fig 4). This position makes it comprehensible why a congenital heart block exists in some cases of congenital malformations of the heart, because not only the anlage of the septum membranaceum, but also that of the bundle may be affected. Whether or not an embryological " dysplasia " is entirely responsible for the malfor-

electrodes are directly applied to the sinus node the electrical events accompanying its excitation can easily be recorded (electrogram of the sinus node). However, this situation no longer prevails with indirect leads from the surface of the body Only when the impulse leaves the sinus node, and, radiating in all directions, spreads over the auricle does the first wave, the auricular or *P wave*, appear in the electrocardiogram It presents a low, upward (positive), often slightly splintered notch (Fig 5) It develops from potentials accompanied by the activation of both auricles [1]

Normally the right auricle which contains the sinus node is excited first and then, after the lapse of only 0 01–0 03 second, the left follows However, this difference in time is too short to be significant

The time which elapses between stimulus formation in the sinus node and the beginning of excitation of the auricles (sino auricular conduction time) amounts to 0 01–0 015 second Since the activity of the sinus node cannot be recorded directly in the electrocardiogram, the duration of the sino auricular conduction can be determined only experimentally The excitation of the auricles is completed when the apex of the P wave appears in the electrocardiogram

After the stimulation of the auricles the wave of excitation passes to the ventricles by means of the atrioventricular system, the second collection of specific fibres

The *atrioventricular system* begins with the atrioventricular node (A–V node, node of Aschoff and Tawara, 1906) It is composed of a small auricular part lying above the atrioventricular septum and a larger ventricular section located below this septum Both parts possess a different histological structure and are sharply defined from each other (Kung) The position of the A–V node in the auricle is easy to determine The location of the mouth of the coronary vein in the right auricle is established, the A–V node begins immediately in front of it, that is, in the posterior inferior section of the interauricular septum (Fig 4) Like the sinus node it also has junctional fibres which spread out in all directions and create connections with the auricular musculature, the junctional fibres spread especially to the right auricle, to the auricular septum, and to the *left* auricle, they accept the wave of excitation coming from the auricle and conduct it to the A–V system The structure of the A V node is quite complicated, histologically it consists

[1] In this book the electrocardiograms which were recorded with the amplifying electrocardiograph are shown as a dark line on a white background (time marking 0·05 second) The electrocardiograms with a light (grey) curve on a dark background were recorded with a string galvanometer (time record, unless otherwise stated 0·04 second)

almost simultaneously Therefore the force with which the mammalian ventricle contracts is entirely different from that of cold blooded animals where the excitation extends gradually affecting one section of the ventricle after another

As long as the excitation proceeds along the rather extended A–V system no alterations of the electrocardiogram are apparent While the bundle of His its branches etc consist of quite large collections of cells their excitation creates potentials which are far too small for registration from the surface of the body For the most part the potentials are short circuited in the interior of the heart For this reason an isoelectric line again is registered in the electrocardiogram after the P wave Only when the excitation has left the A V system and with the aid of the Purkinje fibres has reached the common muscle does one again see waves in the electrocardiogram the ventricular electrocardiogram

From this it follows that the specific tissue forms and conducts stimuli but its activity in the auricle and ventricle is not directly recorded in the electrocardiogram only its results are visible The waves which are evident in the electrocardiogram develop in connection with the excitation of the common muscle and not of the specific fibres

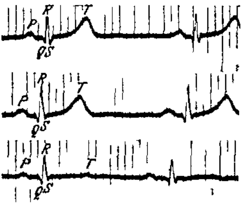

Fig 5 Normal electrocardiogram

The *ventricular electrocardiogram* is composed of two parts *the initial and the terminal deflection* The initial deflection (initial complex or QRS complex) develops from the summation of the single potentials which are formed by the excitation of the ventricles In the classical normal electrocardiogram it consists of a short downward directed Q wave a high upright R wave which is followed by another small downward wave the S wave (Fig 5)

A Q or S wave is never directed upward (positive) and an R wave can never be directed downward (negative) A Q wave must always be located in front of the R and an S wave must always lie after the R Normally the waves are delicate and thin

These designations for the electrocardiographic waves introduced

mation is undecided Fœtal endocarditis may play a part in some cases

Just below the septum membranaceum the bundle divides into two main branches one for the right and one for the left ventricle Each has an entirely different structure The right bundle branch runs as a small self contained cord down to the papillary muscle and only there does it divide into finer branches On the other hand the left bundle branch divides soon after its origin it sends an anterior branch forward a posterior branch dorsally but in between there are also many smaller branches a small branch originating just after the left bundle branch even goes upwards to the musculature of the septum (Mahaim Fig 4)

The main limbs then subdivide into continually smaller branches which form a mesh with each other and finally pass into a network visible only microscopically of peculiar large fibres These invest the inside of the ventricles and convey the transmission of the excitation to the ventricular musculature (Purkinje fibres)

In order for a stimulus to pass from the auricle to the ventricle it must travel along the entire long path just described The specific tissue of the auriculo ventricular system is in contact with the working musculature at only two places The connection with the auricles is accomplished by the ramifications of the A–V node that to the common muscle by the Purkinje fibres

The existence of connecting fibres which are said to run through the ventricular septum and to create a connection between the Purkinje networks of the right and left ventricles has been claimed by Wahlin as well as by Cardwell and Abramson their descriptions still lack confirmation

The specific tissue to be sure lies embedded in the ventricular musculature but is separated from it by a kind of lymphatic sheath If in certain animals a dye is injected into this lymph sac the ramifications of the specific tissues are stained in their totality but the common muscle remains unstained This isolation of the specific tissue in combination with its special blood supply (p 17) results in the fact that the A V system may not necessarily be simultaneously affected by diseases of the common muscle The conduction of stimuli is often normal when the common muscle is severely damaged On the other hand the specific tissue *alone* may be affected despite an intact common muscle *The specific tissue has its own pathology*

The complicated structure of the conduction system permits the excitation to reach approximately all parts of the ventricle

single fibre requires a longer time so that the T wave is broader and more sluggish than the QRS complex

If recovery were to occur with the same speed in every single part of the myocardium and in the same order as the excitation and if the heart did not alter its position in the thorax during systole, the terminal deflection would be the exact mirror image of the initial deflection. Actually, however the recovery process proceeds much more slowly and probably requires different lengths of time in various muscle segments, it proceeds in a *similar* but not in an identical sequence as the excitation, moreover the position of the heart decidedly changes during contraction All these factors are responsible for the fact that the recovery wave the T wave shows an entirely different shape from that of the initial deflection Normally T is a low, positive wave which evolves slowly

Quite frequently in the electrocardiogram of normal individuals another wave, even more sluggish and lower is found after the T wave, it has been designated the *U wave* Since it is located after the T wave, at the beginning of diastole it certainly is unrelated to ventricular excitation or recovery A conclusive explanation for it is not available but the following suggestion seems to be the best The systolic stroke volume of blood is at first received by the large vessels and then advanced The U wave has been ascribed to the excitation of the smooth muscle in the initial part of the aorta and of the pulmonary artery (Hering) The presence or absence of the U wave has no pathological significance (U waves are evident in Figs 8, 9 and 103) U waves are often found especially in electro cardiograms which have been recorded after physical exertion The authors cannot confirm the report of other workers that a U wave can be found in *every* electrocardiogram that has been accurately recorded in all respects

The waves of the normal electrocardiogram in different individuals and in the single leads of the same patient exhibit very decided variations in form which will be considered on p 20

A knowledge of a few numerical values is necessary for the appraisal of the electrocardiogram In the first place, one is interested in the time which elapses between the auricular and ventricular activation (conduction time) In the electrocardiogram it is determined by measuring the distance between the *beginning* of the P wave and the *beginning* of the initial deflection This P–Q interval (in the absence of a Q wave, the P–R interval) embraces the excitation of the auricles *as well as* the conduction of excitation in the A–V system Therefore in cases in which intra-auricular

by Einthoven are generally employed and have resisted all attempts at a new nomenclature But they give rise to several difficulties which should not be left unmentioned

If electrocardiograms are recorded simultaneously in several leads when an R wave is registered in one lead an S sometimes appears in another In this event some authors have designated the S wave as a negative R This procedure has led to confusion and for this reason is not recommended

In dextrocardia the initial deflection in Lead I is the mirror image of the normal (see p 38) even in this case it is not advisable to describe the tracing as though a positive Q wave were followed by a negative R and then again by a positive S wave It is much better to retain the rule and to call the first upward deflection an R wave the downward deflection which precedes it as Q and the downward deflection which follows it as an S wave

Not rarely (with the string galvanometer more frequently than with the amplifying electrocardiograph) a small or large positive wave is found after the S wave (Fig 34) this wave has no name (it has been called the S_2 wave or innominata it has no practical significance) In several disturbances of intraventricular conduction several additional new waves may appear In this event it is advisable to describe the initial deflection in a manner that the direction of the wave is reported as + or − and in addition to mention their amplitude in millimetres (McGinn and White) This method is very useful and deserves wider application in the description of all electrocardiograms not accompanied by tracings It provides an accurate conception of the electrocardiogram which is being described An example of the description of the initial deflection in a normal case (such as Lead II Fig 5) would be − 1 + 11 − ½ By means of this description the direction and height of the waves of the initial deflection is clearly and unequivocally expressed

Formerly the individual waves were associated with the excitation of definite parts of the heart But these efforts were abandoned after it was proven that the excitation reaches almost simultaneously through the conducting system the most diverse areas of the right and left ventricles the region of the apex as well as the base

As soon as both ventricles are activated an isoelectric line or S–T segment (or in the absence of an S wave the R T segment) is registered since at that time no differences in potential exist at the sites of derivation But as soon as recovery from the excitation begins (deactivation) differences in potentials reappear and the terminal deflection *the T-wave* occurs and appears in the electrocardiogram

The excitation process itself evolves very rapidly and produces steep waves of short duration Recovery from excitation in each

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Likewise the *innervation* of the specific tissue is extensive Each specific *element is surrounded by a net* of nerve fibres so that the greatest difficulties are encountered in the experimental separation of the nerve and muscle fibres The nerve fibres belong to the vagus and sympathetic system

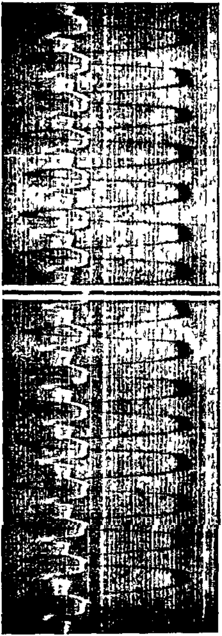

Fig 7 No change of intraventricular pressure during vagus stimulation The cessation of stimulation of the vagus is marked by the white line

Sympathetic fibres can be demonstrated everywhere in the heart *Although very abundant* in the sinus node they are less numerous in the A–V node, but they are also found in the deeper sections of the ventricles Vagus fibres, however, are found with certainty only as far down as the region of the A–V node and have never been seen histologically in the deeper *sections of the ventricle* nor have direct vagus effects been unequivocally proven in the ventricle either by pharmacological or other experimental methods Early works, dealing with this question, arrived at the opposite conclusion, but they have not been confirmed by recent studies performed with modern methods (Drury, Rothberger and Scherf)

Both coronary arteries participate in the nutrition of the left bundle branch, but the right bundle branch obtains its blood exclusively from the Ramus descendens of the left coronary artery Mahaim strongly emphasizes this fact since it creates unfavourable conditions for the nutrition of the right bundle branch

The schematic drawing in Fig 6 (modified from Mahaim) further describes the nutritional arrangements of the A–V system

Numerous variations are found in the course of the vessels of the A–V system Thus in about 10 per cent of cases the left coronary artery

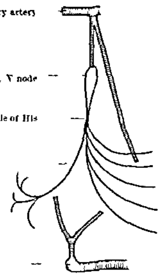

FIG 6 Schematic drawing of the arterial blood supply of the auriculo ventricular conduction system (after Mahaim)

mostly participates in the nutrition of the bundle of His and of the A V node

Abundant anastomoses exist between the different branches of the coronary arteries In those cases of aortitis in which the orifice of one coronary artery is completely obliterated and the other so markedly reduced that the orifice is smaller than the head of a pin we have never seen a disturbance of stimulus formation or of stimulus conduction appear in other words anastomoses of the coronary system with the bronchial arteries or the pericardial vessels, the vasa vasorum of the aorta and the vasa Thebesii may be sufficient for ensuring the compensatory blood supply of the specific tissue

The separate blood supply of the specific tissue and the numerous connections between the branches of the two coronary arteries help to maintain its independence from the working muscle, and tend to

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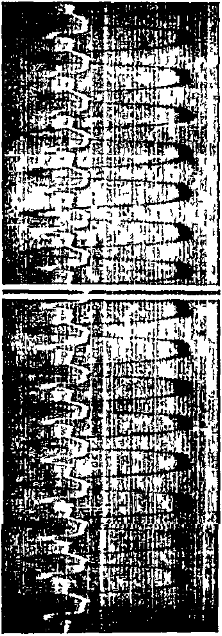

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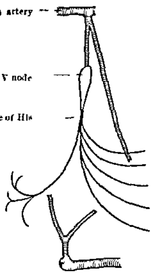

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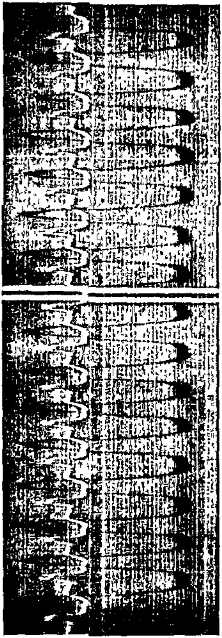

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No proof exists of *direct* vagus action depressing contraction stimulus formation or conduction in the *ventricle* of the mammalian heart Therefore the oft-described vagal cardiac weakness does not exist Considered from the teleological standpoint this situation is very advantageous for the organism it would constitute a considerable source of danger for the survival of the organism if the vagus nerve a depressant of all the fundamental properties of the heart could directly influence the ventricle

The right ventricle of a dog (Fig 7) was rhythmically stimulated with a suitable apparatus so that a ventricular tachycardia appeared The intraventricular pressure was registered in the left ventricle In the illustration the broad white lines near the centre of the tracing indicate the cessation of stimulation with a faradic current of the right vagus nerve The portion of the curve to the right of the white signal lines shows the intraventricular pressure after vagus stimulation remaining the same as the pressure during stimulation It is apparent that the intraventricular pressure does not vary under these conditions Stimulation of one of the sympathetic nerves of the heart for a few seconds considerably increased the intraventricular pressure

To be sure the vagus can *indirectly* alter ventricular activity in three ways

1 Through depression of stimulus formation in the auricle and through the slowing of conduction to the ventricle If this depression exceeds a certain limit the ventricle which is independent from the vagus aids itself by its own automatism

2 Through alteration of the calibre of the coronary arteries which are constricted by the vagus

3 Upon stimulation of the vagus the vagus substance of Lœwi (acetylcholine) liberated in the auricle may reach the ventricle through the venous ostia and coronary arteries and exert a weak effect demonstrable only under special conditions (Scherf confirmed by Engelhart)

In many animals used for experiment the right vagus has a greater action upon the sinus node (stimulus formation) and the left more upon the A-V node (and therefore on stimulus conduction) (Rothberger and Winterberg) In man these differences are usually absent

VARIATIONS IN THE FORM OF THE NORMAL ELECTROCARDIOGRAM

No two people on earth have identical electrocardiograms Every electrocardiogram shows some individual characters Only in

homologous twins are there remarkably great similarities in the electrocardiogram (Almeida Prado)

In Fig 8 may be seen sections of tracings in the three leads from seven patients who had normal circulation and who were free from symptoms The tracings show distinctly the marked differences in the form of the electrocardiogram recorded in various individuals These differences are so great that it has been suggested that the electrocardiogram rather than the finger prints of criminals should be recorded Naturally this suggestion was not adopted because the electrocardiogram can undergo decided and rapid alterations from a variety of causes

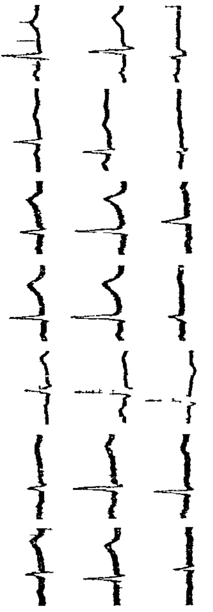

Fig 8 Normal electrocardiograms from seven patients who had no disease of the cardiovascular system The three leads are placed under each other

In the healthy individual atypical waves are most frequently displayed in Lead III which is so often the lead of exceptions that one can rarely obtain the classical normal electrocardiogram in a healthy person *in all* leads In Fig 8 which is not intended to *represent selected tracings* in every case Lead III is atypical in some particular

Even the P wave frequently shows variations in form in Lead III it may be markedly splintered in visible or *negative* Approximately 15 per cent of healthy people show atypical P waves in Lead III

As evidenced by Fig 8 the initial deflection is often splintered

plump or notched in Lead III The T wave may be absent inverted or diphasic These variants in the form of the T wave in Lead III are found in approximately 40 per cent of normal people Even when they are absent at first and appear during observation they cannot be evaluated since—as will be shown later—extreme alterations in the T wave can appear in Lead III as the result of alterations in the position of the diaphragm

The normal range of variations in the electrocardiographic waves is much smaller in *Leads I and II*

Sometimes the *P wave* is pointed often it is split In Leads I or II (less often than in Lead III) of the normal individual it may also be very low or practically invisible It may have a width up to 0 10 second and rarely exceeds a height of 2 mm

The *P Q distance* or interval which follows the P wave frequently lies below the isoelectric line This is the effect of an after deflection which follows the P wave similar to the T wave which follows the QRS-complex The after deflection of the P wave is called the T auricular wave (Ta wave) If the P waves are very large this T of the P can be recognized in the P Q (or P R) segment lying deep below the isoelectric line It can be seen more easily when the P wave is not followed by a ventricular complex (cases of heart block) As a rule this final deflection of the P wave is inverted with a positive P wave and is directed upward with a negative P wave that is they run in opposite directions (Fig 102) If the electrocardiogram is recorded after physical exertion Ta is more marked The T of the P wave (the Ta wave) proceeds so sluggishly that it may even influence the position of the S–T segment (see Fig 198)

The length of conduction time measured from the beginning of the P wave to the beginning of the Q (or R) varies—as mentioned earlier—between 0 12 0 20 second Measurement of tracings may reveal that this distance is not the same in all leads This is because the beginning of the P wave sometimes cannot be determined with exactitude in one or another lead since it does not always rise abruptly from the base line likewise the first part of the initial deflection cannot at times be determined The P waves and the initial deflections like all of the other waves of the electrocardiogram develop as might be anticipated from the sum of positive and negative potentials which if registered individually produce upward *and* downward deflections in the electrocardiogram The summation of equally large positive and negative potentials for a time can yield zero values It is recommended that one should consider the longest

conduction time of the three leads as most closely approximating the correct value

In the healthy individual the length of conduction time may vary *between the limits stated depending upon the* cardiac rate On the average it is shorter in children

The Q wave is often absent Normally it does not exceed a depth of 3 mm in Lead II In exceptional cases the R wave may attain the height of 20 mm the S wave which in many cases is also absent in one lead or another may be twice as deep as the Q wave *Under normal circumstances the R wave is highest in Lead II However exceptions are not rare*

Slight splitting and notching of the initial deflection also occur normally they are found especially often near the base line and are attributed to a congenital abnormality of structure of the conduction system which is phylogenetically recent and shows many variations *The width of the entire initial deflection which is always measured from the beginning* of the Q wave (or in the absence of a Q from the R wave) to the *end* of the S wave (or R wave if there is no S) varies in the healthy individual as stated above between 0 05–0 10 second but it is usually less than 0 08 second On the average the longer intervals are found in tall men of powerful physique For reasons previously cited *it is not equally long in all leads If different* values are recorded in individual leads the highest is taken as approximating the correct value where chest leads are recorded it is usually highest in that lead (see p 150)

As already mentioned the *S–T segment* varies in length In the healthy person it does not always lie exactly on the baseline *In string galvanometer records normally it may lie a few tenths of a* millimetre below or 1 3 mm above the isoelectric line In the amplifying type of electrocardiograph displacement up to 2 mm *above* the isoelectric line is normally possible An over active heart (after physical exertion or hyperthyroidism) may lead to slight downward displacement of the S–T segment (see also p 142)

In Leads I and II the normal T wave is at least 1 5 mm high, it may attain a height of 5 mm and rarely more It is usually rounded, rarely pointed and the beginning and end are not abrupt With a low diaphragm the T wave in Lead I can be very low It is never split or notched and is rarely bifid

It is clear that all numerical values for the amplitude of the waves of a normal electrocardiogram hold only *for those electro* cardiograms which are taken with an apparatus standardized at 1 cm for one million and with the patient in the circuit

Most of the electrocardiograms in Fig 8 show some peculiarity although they were recorded from healthy young people

In the first case (from left to right) the P wave in Lead III is negative and in Lead II it is almost invisible In the second case the P wave shows splintering especially in Lead I and the initial deflection is low in this lead In the third case notching may be seen in the descending limb of the R wave near the baseline and the T wave in Lead III is negative In the fourth series of tracings the S–T segments are distinctly elevated in Leads I and II, the initial deflection is thick (slurred) and notched and the T wave is very small The S–T segment is also definitely above the isoelectric line In the fifth case the T in Lead III is very low In the sixth tracing the initial deflection is abnormal in Lead III and distinct U waves are evident especially in Lead II In the seventh series the P wave is split in all leads, the initial deflection shows a deep split S wave and the T wave is almost invisible in Lead III

DEFORMATION OF THE ELECTROCARDIOGRAM DUE TO UNSUITABLE RECORDING

The electrocardiogram can be deformed by unsuitable connections, for example by polarizing electrodes, or by the use of condensors of unsuitable values in series with the patient so that pathological alterations are simulated Under these conditions some waves may show abnormal magnification or reduction, the S–T segment may be displaced, and the T waves may be diphasic

Fig 9 reproduces two electrocardiograms of the same healthy twenty year old man recorded immediately after each other, the upper tracing was transcribed by means of a large string galvano meter with a condensor in the circuit, the lower tracing with an amplifying electrocardiograph In the string galvanometer tracing a very deep S wave exists in Lead I, while in that of the amplifying electrocardiograph the S wave is much smaller In Leads II and III S waves are recorded only with the string galvanometer while they are absent in the record of the amplifying electrocardiograph

In Lead I of the string galvanometer record the S–T segment is displaced above the baseline in an entirely abnormal manner (effect of polarization) the record from an amplifying electrocardiograph shows a normal S–T segment The T waves in Lead I are also positive in the electrocardiogram of the string galvanometer, but they show a distinct negative after phase which represents a typical

polarization effect, this is absent in the tracings recorded with the amplifying type of apparatus U waves (most distinct in Lead II) are seen in both tracings

It is typical for electrocardiograms recorded in a technically improper manner by the string galvanometer to show too deep S waves abnormal S–T segments and diphasic T waves With accurate technique such features are encountered only under pathological conditions Lack of knowledge of this fact often leads to errors of serious import

Opinions vary on the question of whether the string galvanometer or the amplifying electrocardiograph yields more accurate tracings and which, for this reason should be preferred Among other things it is stated of the string galvanometer that the current flowing through the apparatus leads to a fall of potential This fall

FIG 9 Two electrocardiograms from the same patient The upper tracing was recorded with a string galvanometer the lower with an amplifying apparatus

of potential is questioned by others or considered so slight that it is devoid of significance

However, a disadvantage of some amplifying electrocardiographs consists in the fact that the electrocardiogram is deformed by amplification

The authors are of the opinion that an apparatus constructed in accordance with either principle will yield satisfactory tracings so long as certain rules in the technic of recording are observed (no condensor in the string galvanometer) and the peculiarities of the apparatus concerned are considered

There are several other reasons for the *deformation of the electro cardiogram by external influences*

1 Restlessness of the patient movements of the head or the extremities All of these bring the string out of its zero position through muscle action currents foreign to the heart (Fig 10b) If a tic exists the disturbance recurs at regular intervals so that pathological cardiac waves may be simulated

2 Frequently the electrocardiogram is deformed by *alternating*

currents There are rooms and structures which are haunted by stray induction currents At times these may obscure an electrocardiogram so greatly that the tracing cannot be evaluated This disturbance is recognized by the regularity of the vibrations in the electrocardiogram (Fig 10c) Many of the tracings in this book show vibrations of alternating currents to a slight or marked extent Often this disturbance may be abolished or diminished by wrapping the patient in an isolating cover

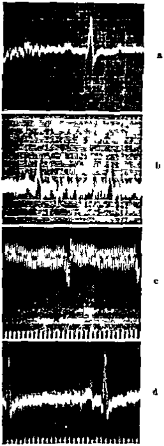

FIG 10a–d Deformation of the electrocardiogram from external shaking (a) restlessness of the patient (b) alternating currents (c) and muscle tremor (d)

3 Not rarely the electrocardiogram shows disturbances as the result of slipshod contact, and this may give rise to confusion with abnormal waves (see Fig 75)

4 Tremor of the patient in hyperthyroidism in the senile, and in the profoundly cachectic individual may induce disturbances which at times make a finer analysis of the tracing impossible (Fig 10d) This change is usually most marked in Lead I, and much slighter in Leads II and III since usually both arms participate in the tremor Sometimes it is necessary to employ a chest lead in order to interpret the electrocardiograms of these patients, particularly with respect to the smaller waves To minimize this disturbance the patient should lie in a warm room the bandages for the application of the electrodes, if no jelly is used must be immersed in a *warm* saturated salt solution, and the muscles should be relaxed as in sleep

5 Shaking of the apparatus (from the slam of a door, the passage of a heavily loaded truck in front of the building in which the electrocardiogram is being taken) can also alter the electrocardiogram (Fig 10a)

EINTHOVEN'S TRIANGLE

The electrocardiogram which is recorded from a patient in the three leads is produced by one and the same event, the activation

and recovery process in the heart Figuratively this event is observed from three standpoints When the heart—the organ providing the electrical potentials—is altered in any way, these changes are expressed in a definite manner in the three leads According to the location and the extent of the alterations in the excitation process, they will be *more* evident in one or another of the leads But since the same event is investigated in all leads, it may be anticipated that definite relations will exist between the three leads in regard to the amplitude and the form of the waves

This relationship was discovered by Einthoven, and is expressed by the simple equation Lead II = Lead I + Lead III

If Leads I and III are simultaneously recorded with an appropriate apparatus (double string galvanometer),[1] see Fig 35, one can reconstruct Lead II from the *algebraic* sum of the waves of Leads I and III If there is a positive P-wave in Lead I and an equally negative P-wave in Lead III, the P wave may be absent in Lead II (see Fig 8, first curve on the left) If a medium sized R wave exists in Leads I and III, the R wave as the exact sum of R_1 and R_3 is highest in Lead II A glance at the electrocardiograms depicted in this book and shown with the three leads will confirm the validity of this rule *An exact reconstruction of Lead II is* possible only when Leads I and III are recorded *simultaneously*, since only under these circumstances can the summation of each phase of the initial and terminal deflections be performed At times the initial deflection rises from the baseline later in one lead *than in another so that one cannot simply summate the apices* of the R– or S–waves An R–wave in one lead may correspond to an S–wave in another, etc

The mathematical proof for this rule was furnished by Einthoven The electrocardiogram is recorded from the extremities, but this *furnishes only the difference of potential which exists at those areas* of the trunk of the body from which the extremities depart When the electrode is placed on the right arm, the same electrocardiogram is obtained as from the right shoulder The arm is merely the conducting medium which, like a wire carries the current to the apparatus *If the areas at which the right and left arms as well as the* left leg arise from the trunk are connected with each other, approximately an equilateral triangle is obtained (Fig 11) Let us assume that the direction of the spread of excitation (the electrical axis)

[1] For experiments of this kind the amplifying electrocardiograph is better than the string galvanometer because in the latter one lead influences the form of the other

forms a definite angle with the horizontal and furthermore that the potential formed in the heart at a given moment has a definite value represented by the line e A definite portion of this potential will be measured in the three leads The amount is obtained if perpendiculars are drawn from the end points of the line e to the three lines *of derivation* *Measurement actually demonstrates that the* projection of e on Lead II is equal to the sum of the projections on Leads I and III ($e_2 = e_1 + e_3$)

*If the direction of the electrical axis changes, alterations imme*diately appear in the different leads even when the potentials of the heart (e) remain the same From the triangular diagram one can

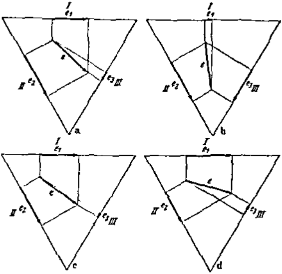

Fig 11a–d Triangle of Einthoven

deduce all of the possibilities met with in actual practice With an almost perpendicular position of the cardiac axis, the projection on I is small (Fig 11b) and it will be shown later (p 42) that in this case the initial deflection in Lead I actually appears very small Likewise when the angle formed between the electrical axis and the horizontal approximates 30 degrees the projection on Lead III is very small (Fig 11c) With still smaller angles a reversal of the direction of the deflection will be found (Fig 11d)

Apart from these geometrical proofs there are also exact calculations which need not be cited here since they have no practical significance They may be read in any of the larger text books of physiology

If an iron plate is cut in the shape of an equilateral triangle and if in *the approximate centre of the triangle electrical potentials of a definite* size and direction are produced and the potentials are led off from the three apices then the correctness of Einthoven's rule can be experimentally proven *The same proof can be obtained in experiments on* the cadaver (Fahr and Weber)

There are small insignificant exceptions to the rule of Einthoven Its practical importance consists in the fact that it readily permits one to decide whether or not the tracings are correctly marked *Errors may occur when fragments cut out of the three leads are* pasted on a card to form a permanent record The leads are correctly marked when they approximately agree with Einthoven's rule namely that Lead II corresponds to the algebraic sum of Leads I and III

METHOD OF ANALYSIS OF THE ELECTROCARDIOGRAM

In every description or interpretation of an electrocardiogram it is recommended that one pursues the following method enabling one to become accustomed to report the individual recognizable points in electrocardiographic tracings in a definite sequence, thus no points are overlooked and the diagnosis is facilitated

1 The rhythm of the heart A distinction is made between rhythmic arrhythmic and allorhythmic cardiac activity An arrhythmia exists when the ventricle beats entirely irregularly an allorhythmia when another rhythm replaces the regular rhythm *that is an arrhythmia is regularly repeated (for example bigeminy* continuous 3 2 block etc)

2 The rate of the heart This should be calculated according to the method given (p 61), often the beginner is deceived by casual *inspection*

3 The form of the P waves in all leads (naturally one always begins with Lead I)

4 The length of the conduction time (from the *beginning* of the *auricular wave to the beginning of the ventricular deflection)*

5 The types of waves in the initial deflection whether a Q an R or S is seen in the different leads and the amplitude (approximately) of these waves

6 *The shape of the waves (whether split thickened or notched)*

7 The S-T interval and the T wave These two sections of the electrocardiogram are usefully considered together because often they cannot be differentiated and in many cases show simultaneous *and similarly directed alterations*

8 Summarization and diagnosis

An example of a complete analysis of a tracing may be given for Fig 5 A regular normal (sinus) rhythm is present The P waves are positive in all leads and slightly split The conduction time (best measured in Lead II) amounts to 0 15 second The initial deflection shows a high R wave in all leads in Leads I and II a short Q and S are also seen The initial deflection is 0 07 second in width and the waves are neither split nor thickened The S–T segment lies at the isoelectric line in all leads the T waves of Leads I and II are upright in Lead III the T waves are almost invisible

There is no evidence of myocardial disease The atypical T wave in Lead III appears quite often in the healthy individual

RESPIRATORY ARRHYTHMIA

The heart of the healthy individual does not beat quite regularly The centre of stimulus formation in the sinus node the normal pacemaker is continually under the influence of the extracardiac nerves Variations in the tonus of these nerves by way of reflexes continually lead to alterations in the cardiac rhythm of healthy persons although they are lying perfectly quiet

The most important and frequent form of sinus arrhythmia[1] is the respiratory arrhythmia It is most marked and very regularly present in children and young individuals but it also occurs in people of advanced age It may be discussed at this juncture since it belongs to some extent to the normal electrocardiogram

Often the respiratory arrhythmia actually is regarded as evidence of a healthy heart (Mackenzie) but this is certainly incorrect It may be encountered in a patient with a mitral or aortic valve lesion as well as in severe atheromatosis or coronary sclerosis Occasionally it is pronounced even in a case of coronary thrombosis But it always vanishes when the least decompensation appears and as soon as the reserve power of the heart is exhausted *The decompensated heart beats as regularly as a clock as long as a normal sinus rhythm exists* In other words the respiratory arrhythmia does not indicate that the heart under investigation is healthy Like decompensation physical exertion and the elimination of vagus action by atropine also abolishes the respiratory arrhythmia It is an interesting fact that it can be removed by any mental exertion even by working an easy mathematical problem

[1] Other forms of sinus arrhythmia for example sinus tachycardia and sinus block will be considered in appropriate sections

The great practical significance of the respiratory arrhythmia consists in the fact that it shows that the heart works without effort *in the beaten track* (Wenckebach) A heart which contracts with perfect regularity during complete rest or deepened respiration is suspect from the start

As a rule in respiratory arrhythmia there is an acceleration of the heart in inspiration and a slowing during expiration Since the change in rate may lag somewhat after the corresponding respiratory phase occasionally there are small shifts At times the arrhythmia may become very marked since the rate may become twice as fast at the height of inspiration as in expiration Not rarely during the examination of these patients physicians are disquieted by the arrhythmia and assume the presence of a serious disease But if these patients stop breathing for a moment the arrhythmia vanishes completely so that the correct diagnosis can be made without an electrocardiogram The respiratory arrhythmia is extremely marked in the dog which has a high vagus tonus (dog pulse) Sometimes the arrhythmia is so slight in man that it can be found only upon measurement of the tracing

Fig 12 Respiratory arrhythmia (Lead II)

The electrocardiogram of a patient with respiratory arrhythmia shows a periodic acceleration and slowing of the heart without other alterations

Fig 12 shows a respiratory arrhythmia in a healthy individual during slow deep respiration The slowing—in the middle of the tracing—coincides in time with the beginning of expiration In inspiration the rate is approximately doubled

The respiratory arrhythmia is certainly dependent upon the vagus According to E Hering it is produced by a periodic increase and decrease of vagal tonus arising from the pulmonary branches of the vagus According to other views it concerns impulses which proceed to the vagus centre directly from the respiratory centre (C Heymans) Experimental denervation of the vaso sensory zones in the aorta and the carotid sinus causes the respiratory arrhythmia to vanish

Marked respiratory arrhythmia is found at times during convalescence from infectious diseases and in compensated cases of coronary sclerosis and atheromatosis, occasionally also following coronary thrombosis

In sinus arrhythmias which are not induced by respiration the suspicion is justified that an organic disease of the sinus node exists (see p 296)

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THE ALTERATIONS OF THE FORM OF THE VENTRICULAR COMPLEX AND THEIR SIGNIFICANCE

THE EFFECT OF CARDIAC POSITION ON THE ELECTROCARDIOGRAM

Electrocardiographic Changes due to Respiration

In many normal persons periodic variations in the form of the waves may be seen *simultaneous with a respiratory arrhythmia* but also independent of it The P waves may grow larger, smaller, even vanish or become inverted The waves of the initial deflections may change in amplitude, some new waves appear and others vanish Likewise a positive T wave may become periodically larger or smaller with respiration and may even become inverted in Lead III Any of these alterations are most evident in Lead III,

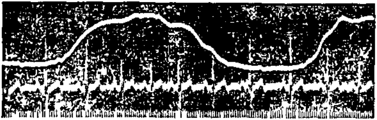

Fig 13 Alteration in the size of the waves of the initial deflection produced by respiration

but they are often at least indicated in other leads As soon as the patient stops breathing, the electrocardiogram remains unchanged in the respiratory phase to which it belongs

Fig 13 illustrates the alterations of the form of the initial deflection dependent upon breathing *The tracing above the electrocardiogram records respiration* In inspiration (upward excursion *of the respiratory curve*) the initial deflections become smaller, in expiration larger The highest R-waves appear at the respiratory pause Slight respiratory arrhythmia also is present (see Fig 35)

These modifications in the form of the electrocardiogram are the

result of changes in cardiac position due to movement of the diaphragm During respiration the heart is more or less displaced and undergoes a slight rotation around its axis In inspiration the rotation is usually clockwise in expiration counter clockwise This rotation is the main reason for the changes in the wave form described

Endeavours have repeatedly been made to ascribe to patho logical changes these alterations in the form of the electrocardio gram which are the result of respiration however no proof has been advanced to substantiate this interpretation

There are cases in which even extreme alterations in the position of the diaphragm or displacement of the heart by an effusion fail to have any marked influence on the electrocardiogram in these instances no rotation of the heart around its axis but simply a lateral displacement had occurred The position of the heart in the thorax (constitutional factors) also affects the extent of alterations of the electrocardiogram in respiration While these modifications have no practical significance knowledge of their occurrence is important since otherwise they may be mistaken for pathological processes Reference will be made subsequently (see p 67) to the fact that a change of position of the heart even in the healthy individual may suffice for the appearance of an inverted T wave and a deep Q wave in Lead III (Figs 35 43) These problems are discussed under appropriate headings

If the diaphragm is very elevated the P waves initial deflections and the T waves may be inverted in Lead III (Bland and White) However the diagnosis of an elevated diaphragm should never be made from the electrocardiogram alone

The Electrocardiogram in Dextrocardia

In the registration of the electrocardiogram one must always be sure that electrodes assigned to a given extremity are correctly applied to that extremity If an error is made in the registration of Lead I by interchanging the electrodes intended for the right and left arms in accordance with the ampere rule inverted deflec tions are obtained since the current flows through the apparatus in the reverse direction Therefore the P waves the initial deflections and the T waves are directed downward Such an electro cardiogram is the mirror image of the normal

It is exactly this type of electrocardiogram that is obtained in Lead I in cases of dextrocardia In this condition contrary to the normal relations the right electrode is nearest to the cardiac apex and the left nearest to the base of the heart In other words if all

the main deflections are inverted in Lead I, at first one might well believe that the electrodes had been incorrectly applied If this has not happened, the existence of *situs inversus* may be assumed Since in dextrocardia (with or without *situs inversus*) *the base of the heart is still cranial and the apex remains downward*, the longitudinal leads are somewhat modified but not in a perfect mirror like manner, Lead I alone presents the characteristic picture If the heart is otherwise normal, in dextrocardia the largest deflections are found in Lead III

Fig 14 was obtained from a healthy youth in whom *situs inversus totalis* existed All of the main deflections are inverted in Lead I *The normal R wave is converted into a deep Q, the normal S wave* corresponds to a low R No characteristic alterations are apparent in Leads II and III In the electrocardiogram reproduced the P waves are also inverted in Leads II and III and the T wave is absent in Lead II However, these two leads (II and III) not rarely

Fig 14 Electrocardiogram in a case of *situs inversus*

are essentially normal While deviations from the normal ordinarily are most common in Lead III in dextrocardia they are found in Lead II

Naturally the electrocardiogram just described is obtained in a pure form only when the heart is healthy, save for the dextrocardia Obviously the electrocardiogram can show extensive alterations as the result of cardiac disease Displacement of the heart towards the right displacement from tumour effusion or cicatrices (*dextro positio cordis*) never results in the modifications described for dextro cardia, for this reason the electrocardiogram can facilitate the differentiation in doubtful cases

The Electrocardiographic Alterations in the Lateral Position and in *Mediastino-pericarditis*

As a rule the electrocardiogram also undergoes very distinct alterations when the patient assumes a lateral position In chang ing from the right to the left lateral position the cardiac apex

may move about 5 cm. Simultaneously with this movement slight rotation of the heart occurs as the result of the fixation of the basal portion of the heart and owing to the convexity of the diaphragm. It is evident from the preceding discussion that this rotation is mainly responsible for the alterations of the electrocardiogram.

If the electrocardiogram of a healthy individual is recorded in the recumbent and in the right and left lateral positions, alterations of the ventricular complexes are sometimes most clearly seen in Lead I and in other cases in Lead III.

Fig. 15 demonstrates the changes in the electrocardiogram of a normal individual. By turning from the right to the left lateral

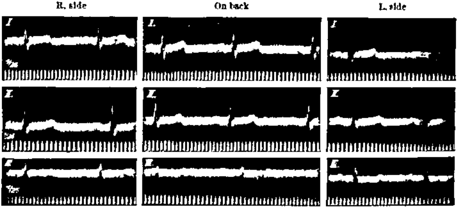

Fig. 15. Alteration of the electrocardiogram in a healthy individual in the lateral position. The three leads are shown under each other.

position the R waves become smaller and the S waves deeper in Lead I. The initial and terminal deflections in Lead III are altered more distinctly (the three leads are placed under one another).

If the heart is fixed by mediastinal adhesions and its mobility thus abolished, the same electrocardiogram is transcribed in each position (Dieuaide). The absence of *rotation* again is responsible. At times X-ray studies demonstrate that some degree of mobility exists although the electrocardiogram indicates a fixation. In this instance it may be assumed that the heart undergoes a lateral displacement without rotation.

Fig. 16 is a reproduction of the electrocardiogram of a patient with adhesive mediastino-pericarditis and shows that no change occurs in the different positions. Rotation of the heart is abolished. The three leads are placed alongside of each other, the upper

electrocardiogram was registered in the right lateral position, the centre in the recumbent, and the lower in the left lateral position

The test is not applicable when the heart is greatly enlarged since the increase in size limits the mobility of the organ

When *concretio cordis* is not accompanied by demonstrable cardiac alterations, the discovery by electrocardiograms recorded in lateral positions of fixation of the heart may be valuable The positive result alone is important

In cases of adhesive pericarditis the QRS complexes and T-waves are often very small (P, D White)

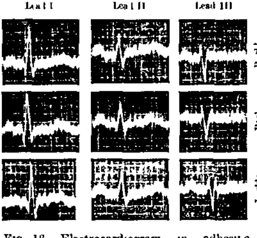

Fig 16 Electrocardiogram in adhesive pericarditis The electrocardiogram does not change at all despite change of position (the three leads are shown alongside of each other)

SMALL DEFLECTIONS IN THE ELECTROCARDIOGRAM

Every wave of the electrocardiogram develops through the summation of numerous potentials which are formed during the activation of the several parts of the heart The P-wave develops from potentials formed in the right and left auricles, the QRS complex and T wave from summation of the right and left ventricular potentials Since the potentials are often directed in opposite directions, the algebraic sum at times yields very small values so that the resulting QRS complex or the P-wave may be very small

To call an excursion small, one must specify the lower limit of normal height These limits are necessarily arbitrary, and quite often there are differences of opinion as to whether or not in a particular case the excursion should be classified " small " In the opinion of some investigators an initial deflection can be designated as low when it does not exceed a height of 5 mm above or below the isoelectric line when recorded with apparatus standardized at normal sensitivity Others regard 6 mm as the limit

Not rarely small excursions occur as the result of technical errors and cause serious mistakes Thus, an undercharged storage

battery may result in small excursions, carelessly applied electrodes may also markedly reduce the excursions in the electrocardiogram

Small Excursions in One Lead

Small excursions in only *one* lead have no practical significance This occurs very often in Lead II, when the direction of the initial deflections in Leads I and III are opposite, for, according to Einthoven's rule Lead II is the sum of Leads I and III If a patient has a positive P wave in Lead I and an equally large negative P wave in Lead III, the P wave in Lead II will be very small or absent The same holds for the other waves (see Fig 7)

If the cardiac axis is more vertical (pendant heart, dropped heart, enteroptosis) the projection on Lead I yields almost a point, as indicated in Fig 11b As would be expected, very small waves are

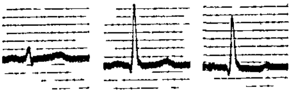

FIG 17 Electrocardiogram in a "dropped heart"

found in Lead I in the conditions mentioned, while the waves closely resemble each other in Leads II and III

However the diagnosis of "dropped heart" should never be made solely on the basis of small excursions in Lead I since the same electrocardiogram also appears for other reasons in patients whose diaphragm is normal in position (very often in incipient hypertrophy of the right heart) (Flaum and Nagel) Initial deflections of this type are not uncommon in mitral stenosis

Fig 17 was recorded in an extremely tall twenty one year old male with a long narrow thorax, low diaphragm and a dropped heart. The ventricular complex in Lead I is very small, while the electrocardiogram is normal in Leads II and III In accordance with Einthoven's rule the waves of the latter two leads appear very similar

If owing to a high diaphragm the transverse position of the heart attains a certain degree, the excursions in Lead III are very small (Fig 11c) and the main deflections in Leads I and II are almost identical (Fig 114)

Small Excursions in All Leads

The amplitude of the electrocardiographic waves depends not only upon the condition of the heart, but also upon its position, and finally upon extracardiac factors such as the condition of the skin and of the tissues surrounding the heart Very low voltage without other alterations may occasionally be found in entirely normal subjects (F N Wilson)

(a) **Pericardial Effusion** A very marked reduction in the size of all waves of the electrocardiogram (low voltage) is found when

a

b

c

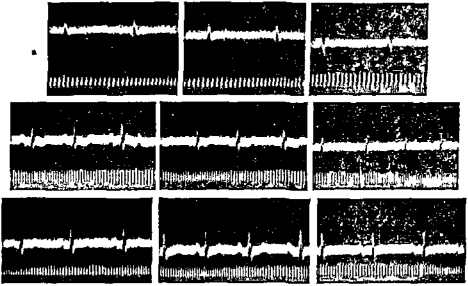

Fig 18a–c The upper series of tracings shows the electrocardiogram in a pericardial effusion and the middle after the removal of 500 c c of fluid *the lower series are the electrocardiograms during the absorption of the effusion*

effusion has occurred into the pericardial cavity (Oppenheimer and *Mann, Scherf*)

Fig 18a shows the electrocardiogram of a patient with a large pericardial effusion The electrocardiogram shows that the smaller waves (P and T waves) have disappeared entirely and the QRS complexes are very low

Fig 18b reproduces the electrocardiogram of the same patient immediately after a pericardial paracentesis by which 500 c c of *serous fluid were removed Although a considerable amount of* fluid remained all the deflections of the electrocardiogram have regained their normal amplitudes The T waves are negative Some

time later (after complete disappearance of the effusion) the electrocardiogram was normal (Fig 18c)

This situation is ascribed to the fact that the mantle of liquid which surrounds the heart short circuits the action currents so that only small potentials are intercepted at the surface of the body The same alterations can be experimentally produced if normal salt solution is injected into the pericardial cavity in this instance the artificial effusion is very small since the normal pericardium cannot be stretched The low voltage vanishes when the effusion is removed by therapeutic measures

The reduction of the amplitude of the excursions is not found in every case of pericardial effusion For example it may be absent when the heart is not *entirely* surrounded by a mantle of fluid and when in some areas the heart is adherent to the pericardium and to the surrounding tissues Alterations of the S T segments and the

Fig 19 The electrocardiogram in myxœdema

T waves in acute pericarditis are described later (p 130) The *small excursions in adhesive pericarditis were mentioned on p 41*

(*b*) Myxœdema A thick dry poorly perfused skin does not conduct currents as well as normal skin In myxœdema the skin may be very thick and dry and a quite characteristic electrocardiogram may be obtained The augmented skin resistance (Zondek) or the heightened capacity of the skin are factors accounting for the modification of the electrocardiogram According to other observers the electrocardiogram of myxœdema is the result of myxœdematous swelling of the myocardial fibres the cardiac enlargement is also attributed by these authors to the same process

In fully developed cases the electrocardiogram shows an extreme reduction of the amplitude of the waves The reduction may be such that the P or the T waves may become almost invisible while the QRS complexes become small and plump and very often attain a height of only 2 3 mm to be sure the changes are not always so extreme sometimes they are absent

The electrocardiogram of a case of myxœdema (in a child) is shown in Fig 19 The P and T waves have practically vanished

and the initial deflections scarcely attain the size of a P wave of a normal electrocardiogram

When the entire symptomatic picture of myxœdema is present, the electrocardiogram is of course unnecessary for diagnostic purposes Nevertheless in obscure cases when an obstinate headache, constipation anæmia or a rough voice suggest myxœdema, the electrocardiogram may provide evidence of diagnostic importance If the patient is treated with preparations of thyroid, the electro cardiographic picture improves simultaneously with the return of normal metabolism

Proof of the fact that the skin alterations are, at least in part, responsible for the abnormal electrocardiogram can be furnished by the fact that if the skin is circumvented by means of needle electrodes

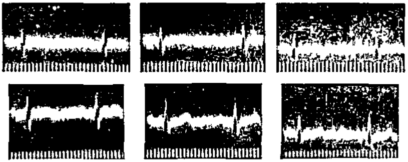

Fig 20a and b Fig 20a (above) shows the electrocardiogram of myxœdema recorded with standard leads Fig 20b reproduces the tracing obtained with needle electrodes inserted beneath the skin

inserted subcutaneously into the proper extremities a normal electrocardiogram is obtained (Nobel and Samet)

In Fig 20a (upper tracing) taken from a seventeen year old girl suffering from myxœdema the P and T waves are remarkably small and the amplitude of the initial deflections is reduced in all leads despite normal sensitivity of the string

Fig 20b was recorded from the same patient the electro cardiogram being recorded by means of needles inserted under the skin of the extremities The skin resistance was avoided through the subcutaneous derivation and an approximately normal electro cardiogram was obtained

Recent investigations tend to refute the assumption that the alterations of the skin are exclusively responsible for the abnormal electrocardiogram In this connexion it should be realized that the amplitudes of the waves in the electrocardiogram of a healthy individual are also greater if subcutaneous leads are employed With

the use of the amplifying electrocardiograph which measures potentials, one might expect that in myxœdema the excursions would not be reduced in size because in this instance the resistance of the skin does not affect the electrocardiogram we have however on several occasions recorded very small excursions in patients with myxœdema despite the use of this type of apparatus

Gordon Freeman and recently Kaunitz have clinically observed cases which are indisputably instances of myxœdema in which the enlarged heart owed its origin to a pericardial effusion It is possible that a pericardial effusion existed in many perhaps in all other cases described under the term large heart of myxœdema In the last three cases of myxœdema with enlarged hearts personally observed this situation actually prevailed While positive histological evidence proving the existence of a myxœdematous alteration of the myocardium has not been reported the small excursions recorded also by amplifying apparatus and the similarity of the electro

Fig 21 Electrocardiogram from a case of coronary thrombosis.

cardiograms in pericardial effusion and myxœdema would find their explanation in an abnormality common to both

In addition to low voltage in cases of hypothyroidism *abnormal or even inverted T waves may frequently be seen in* Leads I and II They may persist for some time even after the initial deflection has regained its normal amplitude after treatment

(c) **Small Excursions in the Electrocardiograms of Decompensated Cardiac Patients** Not rarely small excursions are seen in severely decompensated and œdematous cardiac patients they gradually become larger as the circulation improves and the patient becomes dehydrated For a long time this finding was difficult to understand for otherwise there is no parallelism between the contractile power of the heart and the size of the electrocardiographic excursions in severely damaged hearts

However the extracardiac factors mentioned above can decrease the amplitude of the waves in the electrocardiograms of decompensated patients The dry thickened skin of patients with chronic œdema in conjunction with a hydropericardium can result in small

excursions. It is also probable that the difficulty encountered in obtaining a fœtal electrocardiogram when the electrodes are applied to the sacrum and lower abdomen of a pregnant woman is a result of the fact that the fœtus is to a certain extent "electrically insulated" from the uterus by the amniotic fluid.

(*d*) **Small Excursions in Myocardial Diseases.** Finally, very small deflections (particularly small QRS complexes) are recorded in many patients with severe myocardial diseases, for example, as the result of coronary thrombosis or diphtheria. The cause of the reduction of amplitude is still unexplained. Often the small excursions are not accompanied by any widening or splintering of the initial deflection and only occasionally are they associated with an alteration of the terminal deflection. Moreover, this reduction of amplitude has nothing to do with a diminution of contractility.

Fig. 21 was obtained from a patient who had survived a coronary thrombosis one year before. From a clinical standpoint the patient had recovered completely. The electrocardiogram shows normal P and T-waves, but the initial deflections are remarkably low (see p. 88).

THE ELECTROCARDIOGRAM OF BUNDLE-BRANCH BLOCK

Bundle branch block represents a definite form of disturbance of *intraventricular conduction* which, for didactic reasons, should be discussed at this juncture.

Until recently the presentation of this disturbance was a relatively simple matter since experimental investigation and clinical experience yielded seemingly concurring results. However, in the recent past the bundle branch block problem has been unfolded by new investigations, especially by Wilson and his co-workers. Justified arguments have been advanced against the validity of the old doctrine of bundle branch block; the localization of the disturbance in the heart which for twenty years seemed established in accordance with the old conception has been the subject of controversy; new forms of electrocardiograms produced by block of a bundle branch have been described. Since many still do not regard the new nomenclature and new conception as completely established and retain the old terminology, considerable confusion prevails.

Although the authors consider the objections which have been raised against the old theory as perfectly justified, they are not inclined to regard the problem as completely solved at the present time. For this reason it seems proper to present at first the old classical theory, then to contrast it with the new conception and

finally critically to discuss the arguments for and against each viewpoint

Classical Description

The normal ventricular electrocardiogram develops from the summation of potentials furnished by the right and the left ventricle Accordingly the normal ventricular electrocardiogram is called a *bicardiogram* and develops from a dextro and levocardiogram (Lewis) The potentials of the two halves of the heart are entirely different from each other as might be anticipated from the position and the anatomical structure of the two ventricles

Under morbid conditions it often happens that the pathological process involves one ventricle predominately or exclusively so that the potentials provided by it are changed in respect to their different qualities *This results in a distinct and definite alteration of the* form of the electrocardiogram To appreciate and to understand these abnormal electrocardiograms it is important to become familiar with the electrocardiogram of the right ventricle (dextrocardiogram) *and the electrocardiogram of the left ventricle (levocardiogram)*

This is not easy even in animal experiments, because one ventricle cannot be completely excluded in order to study the potentials of the other

But there is one disturbance in which, at least for a few hundredths of a second only *one* ventricle is activated and thus the potentials of only one ventricle are first recorded in the electrocardiogram If, for example, the right bundle branch is so severely injured by a pathological process that it can no longer conduct (the interruption of conduction is called a " block "), the sole *direct* path to the right ventricle is interrupted At first the excitation reaches the left ventricle in a normal way and only afterwards reaches the right ventricle through the common muscle and the septum Since in these circumstances, *initially* the potentials of the left ventricle alone appear in the electrocardiogram, it is possible to study the waves of the levocardiogram As the result of this disturbance of the spread of excitation still further changes appear in the electrocardiogram which will be subsequently mentioned

The signs of a block of the *right* bundle branch (classical nomenclature) are —

1 The ' left ' form of the ventricular electrocardiogram, the levocardiogram develops through an excitation at first, exclusively limited to the left ventricle Measurements (on the dog heart) have

shown that owing to the block of the right bundle branch the right ventricle is activated 0 03–0 04 second later than normal Since the left ventricle is activated normally through the intact left bundle branch for 0 03–0 04 second the QRS complex is formed only by the excitation of the left ventricle The levocardiogram can be recognized by the fact that in Lead I a high R wave remains (as normal) whereas in Lead III a small R wave is followed by a deep S wave (Figs 22 and 23) It should be emphasized that the occurrence of these waves in the levocardiogram in man depends upon the normal anatomical structure and position of the left ventricle in

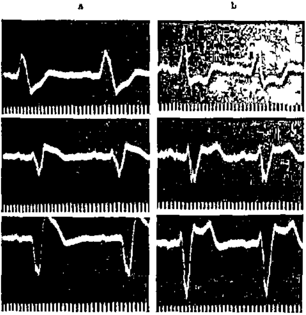

Fig 23a and b *Two cases of bundle-branch block electrocardiograms*

most animals used in experiments even in apes an entirely different levocardiogram is encountered as the result of anatomical peculiarities Lead II, as the sum of Leads I and III depends upon the form of these leads and is not characteristic

2 Normally the ventricles receive the excitation almost simultaneously in their various sections Large potentials develop very suddenly in the heart and immediately afterwards they are neutralized and vanish The QRS complex is completed in a few hundredths of a second But in bundle branch block at first *one* ventricle and then the other is activated, this sequence of excitation of the two ventricles and the slower conduction through the

common muscle must result in a corresponding increase in width of the QRS complex. At times in bundle branch block the initial deflection may measure only 0.10 second. Since in bundle branch block the heart is usually abnormal in other respects additional factors, still to be considered, are present which produce widening of the initial complexes; therefore, in a majority of cases the QRS complex is wider than 0.12 second and often exceeds 0.16 second.

Figs. 22 and 23 show three different electrocardiograms of this type of bundle branch block. In all three curves a marked widening of the initial deflection is apparent. In Fig. 22a it amounts to 0.14, in Fig. 22b to 0.15 second. In Fig. 23 the initial deflection is 0.16 second in width. Moreover a levocardiogram exists in all of these cases and is particularly distinct in Fig. 22a and Fig. 22b. In *clinical* and experimental tracings of bundle branch block a small wave which appears normal (see Fig. 23 in Leads II and III) is frequently followed by a markedly widened one. This widening can appear abruptly and show a step like formation (S step according to Rothberger, Pines).

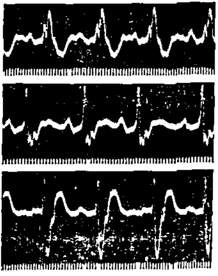

Fig. 23. Bundle branch block.

3. The excitation is normally conducted through the ventricles *with such* velocity and the *activation* is completed so rapidly that the normal initial deflection consists mainly of slender, delicate waves. In bundle branch block, on the other hand, the activation of one ventricle comes *after* that of the other, the stimulus reaching the ventricle whose bundle branch is blocked not through the specific tissue but for the most part through the ventricular muscle which conducts more slowly. Owing to this abnormal spread of excitation and to the gradual successive spread into new areas of muscle, thickening (slurring), notching and splintering of the initial deflections occur. If the ventricular substance is otherwise healthy, the changes may not always be very marked (bundle branch block in animal experiments after section of one branch). If, however, the myocardium is poor or if the smaller branches of the junctional

fibres are also affected, extreme thickening and splintering of the initial deflections may be observed

In Figs 22 and 23 splintering, thickening and notching of the initial deflection may be seen in all the tracings and leads

4 The S–T interval and the T wave are greatly modified Normally, after the QRS complex a section of variable length the S–T segment, occurs in the isoelectric line and is followed by a positive T wave The S–T segment owes its origin to the fact that for a short time after the activation of both ventricles the potentials are balanced If however, there is a block of the right bundle branch, the excitation reaches the left ventricle first and the right ventricle is affected subsequently the activation process has hardly ended on the right side when recovery begins in the left ventricle In other words, the heart has no period of rest and there is no horizontal zero line after the initial deflection the terminal deflection begins immediately at the end of the initial deflection *Therefore its form is modified in a typical way in that the terminal deflection is always directed opposite to the main wave of the initial deflection* For this reason, in a block of the right bundle branch an S–T segment depressed below the zero line as well as an inverted T wave is found in Lead I and an S–T segment displaced above the isoelectric line with a positive T wave is noted in Lead III Moreover, since the S–T interval merges with the T wave, the S–T and the T wave cannot, as a rule, be distinguished from one another, or such distinction is possible only with difficulty

The alteration of the terminal deflection is also the result of the excitation of the two ventricles taking place successively and not simultaneously, as is the case in the normal heart In block of a bundle branch just as we register for a time at least, the initial deflection of only *one* ventricle so the terminal deflection at least its final segment is produced by the recovery of only *one* ventricle (Wilson and Herrmann) In the normal electrocardiogram, a high R wave is followed by an *upright* T wave, the initial and terminal deflections thus having the same *direction* *This is the result of the fact that the T waves, like the waves* of the initial deflection develop through the *summation* of oppositely directed potentials of *both* ventricles Normally, positive values predominate in the sum But if one ventricle is excited *after* the other, the initial and terminal deflections of the ventricular complexes have opposite directions In the discussion of the electrogram of a simple muscle strip (p 2) it was stated that the wave of recovery is opposite in direction to the wave of activation (Fig 2)

If a block exists in the *left* bundle branch, the right ventricle is excited first and a dextrocardiogram is registered by the electro

cardiograph This may be recognized by the fact that *the initial deflection shows a deep S wave in Lead I and a high R wave in Lead III* that is the direction of the waves is the reverse of that of a levocardiogram The widening thickening and notching of the initial deflection are also present In other words if in a right bundle branch block Leads I and III were interchanged one would obtain the electrocardiographic picture of a left bundle branch block (classical nomenclature)

Fig 24 shows an electrocardiogram of this type The initial complexes in Lead I exhibit a short R wave followed by a deep S wave and in Lead III a high R wave is preceded by a short

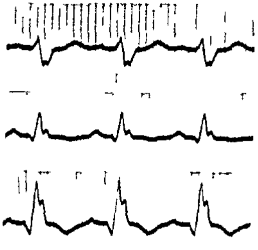

FIG 24 Bundle branch block

Q wave The initial complexes are widened to 0 14 second the waves are somewhat thickened and notched The terminal deflections (S–T interval and T wave) are opposite in direction to the main deflection

This picture of left bundle branch block (classical nomenclature) however is rare it is in fact much less usual than the picture of right bundle branch block described above An explanation for this difference is provided by anatomical considerations For a considerable distance the right bundle branch remains a small undivided bundle and is thus more easily interrupted *in toto* by a pathological process than the left bundle branch which rapidly spreads out like a fan Moreover as a rule the left bundle branch is nourished by two arteries (arising from the right and left coronary

arteries) while the right bundle branch is usually supplied only by the left coronary artery, which is much more frequently affected than the right

Very often the amplitude of the waves of the initial deflection (the "large excursions") is stated as a sign of bundle branch block If it is true that the normal electrocardiogram develops through the summation of potentials of *both* ventricles and that these potentials are usually oppositely directed and consequently *partly abolished by summation, larger deflections would have to be expected* in cases of bundle branch block since at first only one ventricle takes part in producing action currents This is true in many cases However, the factors mentioned on p 43 often play a part and reduce the amplitude of the deflections and, since when patients present bundle branch block the heart is often severely damaged in other respects, deflections of normal size *or even relatively small excursions* may appear

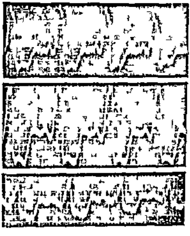

FIG 25 Bundle branch block electrocardiogram with the initial deflections directed upward in all leads

The description which has been given up to this point presents the classical picture of bundle branch block as it has been known since described by Eppinger and Rothberger (1909), Carter and others But deviations from this classical picture at times occur Occasionally *in bundle branch block the direction* of the chief excursions of the initial complexes may be the same in all leads, a common event in many animals used in experiments When confronted with such a situation it was recommended to make the diagnosis from *Lead III, that is, with a deep S wave in Lead III, together with* the other signs of a block, to assume a lesion of the right bundle branch (Rothberger) But objections to this interpretation have been raised and others assert that the direction of the deflection in Lead I should be the guiding principle (Wilson, C J Storm) Since adequate histological controls are not available a decision cannot be made with certainty, but the second proposal seems to possess greater support

Fig 25 shows a bundle branch block with initial deflections which are all directed upward They are 0 14 second in width

and splintered the terminal deflections are depressed deep below the isoelectric line According to the direction of the main deflection in Lead I one may assume the existence of a block of the right bundle branch

Great difficulties are presented by the fact that in cases of myocardial disease without a block of a main stem of the bundle electrocardiographic pictures can be obtained which approximate those of bundle branch block (see p 88) It will be shown later that a levocardiogram widening of the initial deflections and alteration of the S-T intervals and the T waves as in bundle branch block is possible without an interruption of conduction in the main stem of a bundle branch and is even a frequent occurrence in cases of hypertension and coronary sclerosis In these cases it has often been assumed on the basis of the electrocardiogram that a right bundle branch block existed although subsequent histological examination failed to reveal any evidence of disease of this bundle branch as a matter of fact numerous foci may be found in the ramifications of the left bundle branch but no complete interruption of the entire branch is present Myocardial diseases especially coronary sclerosis which is so common in the adult usually and chiefly affect the left ventricle so that such findings are comprehensible

New Nomenclature

Histological evidence of injuries in the region of the ramifications of the left bundle branch has been found in cases which have shown the electrocardiogram of right bundle branch block (Oppenheimer and Pardee) Moreover occasional observations on the appearance of ventricular extrasystoles artificially produced in the human heart exposed at operation (Barker Macleod and Alexander) and finally observations and calculations based on tracings recorded in man and animal experiments have caused Wilson and co workers to diagnose left bundle branch block where formerly right bundle branch block has been diagnosed and *vice versa* A new nomenclature of bundle branch tracings has been created in contrast to the old classical terminology There has been a transfer perhaps prematurely, of the results of animal experimentation to man According to the new conception left bundle branch block would not be unusual on the contrary it would be a rather common occurrence Since no *critical* evidence for these new conceptions has been advanced one opinion is urged against another and great confusion exists Anatomical investigations as far as they have been reported by experienced investigators have supported the

old nomenclature since cases presenting the electrocardiogram of a right bundle branch block (according to the old classical nomenclature) actually have frequently shown a lesion in the right bundle branch (Kauf, Mahaim) On the other hand the results of experiments on the localization of ventricular extrasystoles are definitely in favour of the new nomenclature (p 172) Unfortunately only relatively few cases of bundle branch block have been carefully investigated from an anatomical standpoint so that conclusions rest upon uncertain ground Furthermore as mentioned earlier lesions are in most cases found in both branches Finally, even if the histological examination does not reveal any pathological change this does not prove that during life the bundle branch was capable of normal conduction, since it is known that certain types of myocardial lesions exist without being demonstrable by the histological methods at present available

The new "nomenclature" proposed by the Wilson school was accepted somewhat reluctantly since it implied that right bundle branch block is rare while left bundle branch block is very common This assumption was not in accordance with anatomical facts which made it readily comprehensible why the right bundle branch can be more easily affected than the left

Fig 26 Unusual form of right bundle branch block

However, Wilson and his co workers subsequently published tracings of two "unusual" types of right bundle branch block which actually are not very rare and which permit in harmony with anatomical facts the diagnosis of right bundle branch block with appropriate frequency

One of these types will be discussed on p 58 An instance of the other is depicted in Fig 26 In this tracing it will be observed that there is a low initial complex in Lead I and deep waves in Leads II and III In Lead I of this case the very low R wave is wide splintered and is followed by a short downward directed wave (S wave) In the second and third leads deep S waves are present The width of the QRS complexes is about 0 16 second and the

final deflection is directed opposite to the main deflection On the basis of precordial leads these tracings are regarded by Wilson as representative of a right bundle branch block However if one compares the tracings of Fig 26 with those of Figs 22 and 23 no marked differences will be observed

In the clinic the electrocardiographic diagnosis of bundle branch block is more difficult than in animal experimentation since in the former the situation is much more complicated In experiments it is easy to produce an isolated interruption of conduction in the right bundle branch But in the clinic as the recent investigations of Mahaim have shown usually other foci of disease are present in addition to the injury of the right bundle branch and naturally these exert a marked influence on the electrocardiogram The most common cause of bundle branch block is coronary sclerosis If an occlusion occurs in the descending ramus of the left coronary artery which is most frequently affected not only does the right bundle branch suffer but the anterior part of the left bundle branch also participates This must exert a decided influence on the progression of the excitation and likewise on the appearance of the electrocardiogram

Moreover the direction and the size of the waves is greatly affected by the position of the heart (see p 28 and p 37) The laws which have been established for the normal electrocardiogram also hold for the electrocardiogram with disturbances of intraventricular conduction Thus cases are known in which the picture of left bundle branch block appeared during inspiration and the features of right bundle branch block occurred during expiration (Winternitz) in other cases the change from the right to the left lateral position alters the direction of the deflections in that in the right lateral position a right bundle branch block appears while in the left lateral position a left bundle branch block is evident (Kissin Ackerman and Katz)

In Fig 27 the electrocardiographic signs of right bundle branch block are evident in Leads I and II However in Lead III the electrocardiogram displays the typical form only during inspiration in (normal) expiration an R wave which is neither widened nor notched appears in place of the broadened and splintered S wave

A final and very important difficulty is created in the diagnosis of bundle branch block by the appraisal of the width of the initial deflection The duration of normal initial deflections does not as a rule exceed 0 08 second but in rare cases it may reach 0 10 second The most diverse diseases of the myocardium without bundle branch block may produce widening of the initial deflection

to 0 09 0 10 second or more as well as an alteration in the terminal deflection similar to that of bundle branch block (an S–T segment and a T wave which is pointed in a direction opposite to the initial deflection) On the other hand several cases of bundle branch block confirmed by histological examination have been described in which the initial deflection did not exceed 0 10 second (indeed even 0 09 second) (Mahaim) As a matter of fact the pure bundle branch block of animal experiments fails to disclose the marked degree of widening of the initial complex so frequently found in patients If the existence of bundle branch block were assumed in all curves displaying a levocardiogram initial deflec

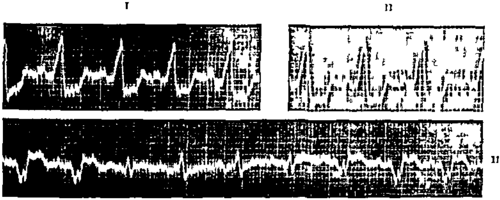

FIG 27 Bundle branch block electrocardiogram with marked alterations in the form of the ventricular complex due to respiration (Lead III)

tions measuring 0 10 second and oppositely directed T waves this diagnosis would be too often made

The initial deflection is usually wider in bundle branch block Often it is 0 14–0 16 second indeed a width of 0 20 second has been observed But this marked widening is partly due apart from bundle branch block to the presence of other foci of disease in the conduction path and in the myocardium Likewise marked widening can certainly occur without blockage of the main bundle

In view of these difficulties and with due regard for the status of our present knowledge it seems advisable merely to state that a tracing shows " bundle branch block type To this statement should be added the fact that it is the common or the uncommon type The common type of bundle branch block corresponds to right bundle branch block according to the classical nomenclature or left bundle branch block according to the new terminology and vice versa Localization of the pathological process with certainty, the demonstration of whether the block exists

in the right or the left branch is, at present, impossible even if it is conceded that the splendid work of Wilson has advanced numerous and strong arguments to support his new terminology *At present it is not possible to assert with complete assurance that a bundle branch block exists at all* It may always involve a levocardiogram whose initial and terminal deflections are modified by intraventricular disturbances of another variety Tracings and transitional curves of this kind are shown in Fig 47 (No 5) and discussed on p 88

It may be expected that further investigations in the near future will enable one to make a more exact diagnosis At the present time the problem of the diagnosis and localization of bundle branch block is one of the most difficult and urgent problems of electrocardiography *Whatever the ultimate explanation may be in each case there is a disturbance of intraventricular conduction, pathological foci are present in the myocardium* (specific tissue) For the clinical appraisal of the case the difficulty of establishing the *site* of the disturbance at present, scarcely has significance

A Special Form of Block of the Right Bundle-branch

Recently Wilson and co workers have described an entirely different picture as the electrocardiogram of a block of the right bundle branch

Fig 28 shows three typical examples In all of the curves the initial deflection is widened to 0 13 second In Lead I the R wave

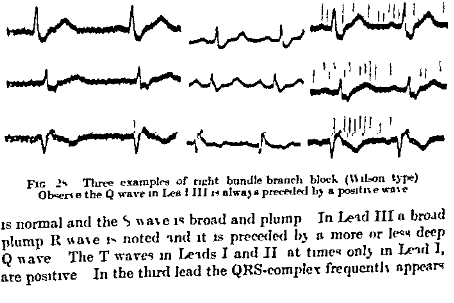

FIG 28 Three examples of right bundle branch block (Wilson type) Observe the Q wave in Lead III is always preceded by a positive wave

is normal and the S wave is broad and plump In Lead III a broad plump R wave is noted and it is preceded by a more or less deep Q wave The T waves in Leads I and II at times only in Lead I, are positive In the third lead the QRS-complex frequently appears

somewhat different. Lead III is as frequently abnormal in this disturbance as in the normal electrocardiogram (Fig. 29) In these tracings the Q-wave is preceded by a small upright wave, therefore it is not a Q-wave in the true sense of the definition of that wave.

On the basis of precordial leads and dog experiments, Wilson and his co-workers assume a block of the *right* bundle Here also the histological proof in man, the crucial evidence as to whether it is a lesion of the right bundle-branch *alone* or in combination with other lesions is still lacking. Nevertheless it must be conceded that *such tracings are commonly observed* and that they are always identical in composition (just as the tracings in Fig 28 resemble each other), so that a definite anatomical substratum must exist

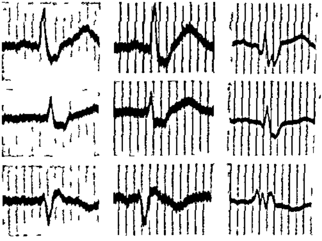

FIG 29 Three cases of right bundle branch block (Wilson type)

In all of these curves it is typical in Lead I for a slender, thin R-wave to be followed by a broad S-wave and in Lead III a slender Q-wave to precede a broad R-wave The T-waves are positive in Leads I and II, negative in Lead III. As a rule Lead III is the mirror image of Lead I *Until histological examination is available, one may call* these electrocardiograms "Wilson block." In regard to the results *of histological examination, it must be emphasized that only positive* findings can be utilized

Fig 29 shows the electrocardiograms of three additional cases with this common type of tracing. The similarity of the tracings is remarkable. If there had been a smaller R-wave and a deeper S-wave in Lead I together with a shorter Q-wave and a higher R-wave in Lead III, the electrocardiogram would look like Fig. 24,

a case of the uncommon type of bundle branch block (left bundle branch block old nomenclature right bundle branch block new nomenclature) Every transition may be observed

Fig 30 shows two more tracings of bundle branch block In the tracings on the left the slender R wave in Lead I and the deep Q wave in Lead III are very long In the series on the right it will be noted that the same waves in another case are very short so that the tracing closely resembles Fig 24 (uncommon type of bundle branch block)

The objection may be raised that tracings of this type are due to a combined lesion in several branches of the conduction system

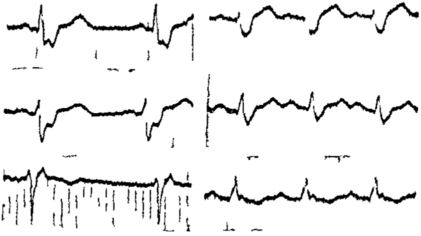

FIG 30 Two cases of Wilson block with transition to the uncommon type of bundle branch block

Against this assumption is the observation that frequently these features develop very suddenly and always display the same alterations of the electrocardiogram (Figs 28–30) we have observed those changes even limited to a few beats (partial bundle branch block see p 62) this would not be possible if abnormal conduction through more than one branch were necessary for producing these features (Figs 32 204)

Not rarely these electrocardiograms are observed after coronary thrombosis (descending branch of the left coronary artery) According to prevailing opinion which the authors do not share the disturbance is one permitting a favourable prognosis The prognosis depends—as always in electrocardiography—upon the underlying disease

If these tracings appear in a case of coronary thrombosis, the electrocardiographic picture persists and does not exhibit the evolution of the alterations in the form of the terminal deflection otherwise observed in coronary thrombosis

It is typical for these tracings to display a thin narrow wave *followed by a very broad one* Thus, a change appears which would actually be anticipated in every bundle branch block and which also occurs in experimental block, in these circumstances first one ventricle is activated with normal rapidity by means of the healthy bundle branch *and only subsequently do the potentials of the* other ventricle develop as the result of excitation over the abnormal pathway In the "Wilson block" electrocardiogram the T waves are directed opposite to the main deflection of the initial complex

The frequent occurrence of low ventricular complexes is unexplained

Wilson recently expressed the opinion that in practically all cases in which the QRS interval measures 0 12 second or more and in which there is a conspicuous S deflection in Lead I the precordial electrocardiograms are of the same type and indicate the presence of right bundle branch block When the QRS interval measures 0 12 second or more, and there is no conspicuous S wave in Lead I, the precordial electrocardiograms indicate the presence of left bundle branch block In both types of bundle branch block the ventricular complex itself is extremely variable in form The only distinguishing feature in the limb leads is the conspicuous S in Lead I in tracings of right bundle branch and its absence in left bundle branch block *The T waves are not necessarily in an opposite direction to* the initial complexes

Significance of Bundle-branch Block

If the electrocardiographic picture of a bundle branch block is obtained, this merely permits the conclusion that a morbid focus exists at some place in the specific tissue This constitutes a valuable discovery, since in doubtful cases it establishes the presence of an organic myocardial disease But the question of *the nature* of the myocardial damage can only be determined from the complete clinical picture, due attention being paid to the history and the other findings, it can never be answered by the electrocardiograph alone A focus of myocarditis, the sequelæ of a coronary sclerosis or thrombosis *invasion by tuberculosis or carcinoma represent* possible lesions The disturbance may be recent, very acute and progressive, but a simple scar may be provocative

If a bundle branch block appears in a case of endocarditis in the course of diphtheria or during rheumatic fever such occurrence proves the participation of the myocardium, if a bundle-branch block is found in a patient suffering from hypertension or angina pectoris the presence of coronary artery disease may be assumed

The clinical diagnosis of bundle branch block is not possible without an electrocardiogram There is however *one* clinical symptom which appears frequently in bundle branch block and this is gallop rhythm According to P D White 38 per cent of the cases of gallop rhythm studied by him had a disturbance of intraventricular conduction The occurrence of this sign precisely in bundle branch block is still unexplained It has been asserted that gallop rhythm develops as the result of non simultaneous contraction of the ventricles but since the difference in time amounts only to about 0 04 second this explanation does not demand serious consideration (Rothberger) Such minute differences in time cannot be detected by the ear It is conceivable that both the bundle branch block and the gallop rhythm are the result of myocardial disease and thereby are not causally interdependent Against this explanation may be urged the fact that when the block appears transiently and persists for only a few minutes gallop rhythm may come and go with the block Accordingly a connection cannot be denied

Owing to the slight difference in time between the contraction of the two ventricles in bundle branch block the significance of this occurrence for the dynamics of the heart is disputed The alteration of the spread of excitation caused by the presence of bundle branch block does possess importance in respect to the affected ventricle Normally the excitation reaches *all* sections of the ventricle almost simultaneously through the ramifications of the A–V system When bundle branch block appears the ventricle supplied by the affected bundle branch is not activated by the rapidly transmitting conduction system but through the slower acting ventricular muscle This does not fail to influence the force of contraction If by a suitable experimental arrangement the intracardiac pressure is registered so that the effect of the normal and abnormal spread of activation can be observed the pressure is actually lower with abnormal excitation

Partial Bundle branch Block

In many publications and books atypical tracings are stated to be indicative of *partial bundle branch block* In this connection it may be pointed out that a disease of a bundle branch which slows

conduction in that branch for only 0 04 second produces the same electrocardiogram as a complete bundle branch block, since in this instance the excitation reaches the affected ventricle *via* the detour from the other ventricle Accordingly one cannot differentiate between a bundle branch block depending upon this delay of conduction and a block resulting from complete interruption In certain rare cases of transient bundle branch block in which the electrocardiogram returns to normal after a few beats or days presumably no complete interruption of conduction was present and simply delayed conduction was responsible

An example of this condition is provided by Fig 31 The electrocardiogram was obtained from a young individual suffering

FIG 31 Partial (intermittent) bundle branch block

from hypertension (280/140) who complained of temporary palpitation After one or two normal beats with slender initial deflections of normal width one or two abnormal QRS complexes may be seen which measure 0 15 second and are followed by abnormal T waves This variation also occurred during rest and without apparent precipitating factors Periodically for one or two beats the conduction system did not function in a normal manner Since it cannot be assumed that *several* branches periodically and simultaneously became incapacitated and since the disturbance must thus be localized in *one* branch electrocardiograms of this type reveal the shape of the initial deflections when *one* branch of the intraventricular conduction system is blocked This variety of proves that in a disturbance of conduction in *one* b conduction system the initial deflection may be 0 15 s

On some days intraventricular conduction was norm

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On some days intraventricular conduction was normal on others

can be easily demonstrated by measurement that this is the result of the algebraic sum of the dextro and levocardiograms (Wilson and Herrmann)

Fig 33 was obtained in an experiment performed upon the exposed heart of an anæsthetized dog After preliminary treatment with barium and mechanical excitation of circumscribed *areas of the right and left ventricles two centres of stimulus formation* were produced they worked with approximately the same rate and alternately controlled the heart At the beginning of the tracing (Lead III) the excitation arises in the left ventricle and a plump widened initial deflection a deep S wave and an oppositely directed terminal deflection is noted Gradually the control passed from the left centre to the right centre (high R wave a plump widened initial deflection and an oppositely directed terminal deflection in Lead III) However when the excitation develops

FIG 33 Gradual transition of an experimentally produced levocardiogram into a dextrocardiogram a bicardiogram in the centre (Lead III)

simultaneously in the left and right centres the ventricular complex is normal in appearance (sixth beat from the left) and measurement reveals that it corresponds exactly to the algebraic sum of the complexes at the beginning and the end of the curve

Experiments of this kind prove that the potentials which are provided by the right and left ventricles give rise to entirely different records and *their sum yields the normal ventricular electrocardiogram*

In the discussion of the electrocardiogram of bundle branch block reference was made to the fact that the levocardiogram is characterized by a high R wave in Lead I as well as a small R wave with a deep S wave in Lead III, in a dextrocardiogram a deep S wave exists in Lead I and a high R wave in Lead III

If in a patient hypertrophy of the right or left ventricle develops one may expect alterations in the electrocardiogram since those potentials which are provided by the hypertrophied ventricle will predominate With increasing hypertrophy of the right or left ventricle the S and R waves in Leads I or III respectively become

Fig 32 Sinus bradycardia with 2 1 bundle branch block

continually abnormal Whenever the conduction in the bundle branch was momentarily disturbed, the heart sounds were distinctly duller than the sounds of the normally conducted beats

Fig 32 shows regular sinus bradycardia of 37 beats per minute (see p 299) Every second sinus beat is conducted abnormally (aberrantly) and a ventricular complex of the bundle branch block type (2 1 bundle branch block) appears One bundle branch conducted only alternate sinus impulses to the ventricle At times especially when a higher ventricular rate was induced (after knee bending exercises) continuous bundle branch block appeared, on many days the electrocardiogram during rest was entirely normal

A partial bundle branch block in the sense that not the entire cross section of the bundle branch is affected but the disease is limited to a part of it does not exist According to the laws of auxomeric conduction in the heart (p 274) such a form of bundle branch disease will not produce electrocardiographic phenomena It is therefore incorrect to designate every tracing with some splitting and widening of the initial complexes as indicating incomplete bundle branch block

RIGHT OR LEFT AXIS DEVIATION (DEXTRO- AND LEVOCARDIOGRAM)

The Significance of Hypertrophy of the Right or Left Ventricle

The normal ventricular electrocardiogram is formed by the summation of potentials of the right *and left ventricles This can be proven by a simple* experiment on the mammalian heart in which the excitation is caused to arise first in the right and then in the left ventricle and the resulting dextro and levocardiograms are recorded Provision must be made for the excitation of the two ventricles to take place simultaneously at certain moments a normal electrocardiogram is then obtained, and it

can be easily demonstrated by measurement that this is the result of the algebraic sum of the dextro and levocardiograms (Wilson and Herrmann)

Fig 33 was obtained in an experiment performed upon the exposed heart of an anæsthetized dog After preliminary treatment with barium and mechanical excitation of circumscribed areas of the right and left ventricles, two centres of stimulus formation were produced, they worked with *approximately the same* rate and alternately controlled the heart At the beginning of the tracing (Lead III) the excitation arises in the left ventricle and a plump widened initial deflection a deep S wave and an oppositely directed terminal deflection is noted Gradually the control passed from the left centre to the right centre (high R wave a plump, widened initial deflection and an oppositely directed terminal deflection in Lead III) However, when the excitation develops

Fig 33 Gradual transition of an experimentally produced levocardiogram into a dextrocardiogram a bicardiogram in the centre (Lead III)

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Experiments of this kind prove that the potentials which are provided by the right and left ventricles give rise to entirely different records and *their sum yields the normal ventricular electrocardiogram*

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If, in a patient, hypertrophy of the right or left ventricle develops, one may expect alterations in the electrocardiogram since those potentials which are provided by the hypertrophied ventricle will predominate With increasing *hypertrophy* of the right or left ventricle, the S and R waves in Leads I or III respectively become

or, more commonly, a levocardiogram. Some patients have a deep S wave in Lead III only during inspiration (presenting the picture *of a levocardiogram), and others* exhibit a dextrocardiogram in one phase *of respiration and a levo* cardiogram in another

Fig 35 was obtained from a case of polycythemia whose heart seemed entirely normal The electrocardiogram was recorded with an Einthoven double string galvanometer The upper tracing represents Lead III, the lower, Lead I Even during quiet breathing the electrocardiogram undergoes continual alteration At the beginning of the tracing during inspiration a deep S wave may be seen in Lead I and a high R wave *in Lead III, in other* words a dextrocardiogram (clock *wise rotation) while immediately* afterwards, during normal expiration, a high R wave occurs in Lead I and a deep S wave in Lead III, that is, a levocardiogram These changes were constantly as evident as indicated in the tracing With a voluntary arrest of breathing the electrocardiogram ceased to show these variations

Coincident with these distinct alterations in the form of the initial deflection, changes also occur *in the terminal deflection* These are especially obvious in Lead III *where an inverted* T wave may be seen at the height of inspiration while it is suggestively positive at the height of expiration

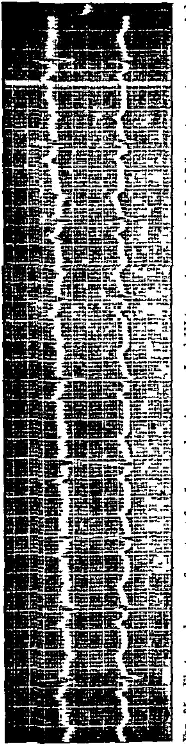

Fig 35 Electrocardiogram of a patient free from cardiac disease Lead III (upper tracing) and Lead I (lower tracing) were recorded simultaneously A dextrocardiogram is seen in inspiration *a levocardiogram in expiration*

higher or lower until a fully developed dextro- or levocardiogram results

In Fig 34a in Lead I there is a deep S-wave, in Lead III a high R-wave (dextrocardiogram) Otherwise the initial and terminal deflections are normal The tracing was obtained from a patient with mitral stenosis whose compensation was perfect.

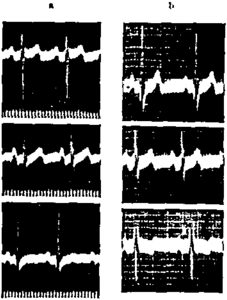

Fig 34a and b Right (a) and left (b) axis deviation in the electrocardiogram

In Fig. 34b the R-wave in Lead I is very high, the S-wave in Lead III very deep (levocardiogram). Similarly, in this electrocardiogram the initial and terminal deflections are otherwise normal. The electrocardiogram was obtained from a patient with a moderately severe, compensated aortic stenosis.

Significance of the Position of the Heart

On the basis of observations of this kind, it was formerly considered correct to present these tracings as evidence of cardiac hypertrophy and it was deemed permissible to diagnose hypertrophy of a ventricle from the electrocardiogram alone Later, it became apparent that quite identical pictures can be obtained in the absence of cardiac hypertrophy especially *when the heart was slightly rotated around its axis*

If, after opening the thorax, the heart of a dog is rotated clockwise around its axis, the picture of a dextrocardiogram is obtained; with counter clockwise rotation a levocardiogram appears (Cohn and Raisbeck) The amount of rotation of the heart required to produce the electrocardiographic changes is so slight that it may be assumed to occur in man commonly Earlier (p. 37) the marked alterations of the electrocardiogram following changes in the position of the heart as the result of deep breathing were described. To these statements may be added the fact that normal people, that is, those without striking alteration of the position of the diaphragm, may frequently be encountered who present a dextrocardiogram

or, more commonly, a levocardiogram Some patients have a deep S-wave in Lead III only during inspiration (presenting the picture of a *levocardiogram*), and others exhibit a dextrocardiogram in one phase of respiration and a levocardiogram in another

Fig 35 was obtained from a case of polycythemia whose heart s e e m e d entirely normal The electrocardiogram was recorded with an Einthoven double string galvanometer The upper tracing represents Lead III, the lower, Lead I. Even during q u i e t breathing the electrocardiogram *undergoes continual alteration* At the beginning of the tracing during inspiration a deep S wave may be seen in Lead I and a high R wave in Lead III, in other words a dextrocardiogram (clockwise rotation), while immediately afterwards, during normal expiration, a high R wave occurs in Lead I and a deep S wave in Lead III, that is, a levocardiogram These changes were constantly as evident as indicated in the tracing With a voluntary arrest of breathing, the electrocardiogram ceased to show these variations

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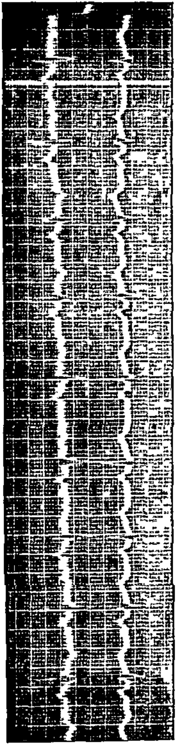

Fig 35 Electrocardiogram of a patient free from cardiac disease Lead III (upper tracing) and Lead I (lower tracing) were recorded simultaneously *A dextrocardiogram is seen in inspiration a levocardiogram in expiration*

Alterations of the same kind but taking place more slowly may be observed as the result of an abnormal position of the diaphragm arising from other factors (distention of the stomach or intestines, pregnancy).

Observations of this kind indicate that it is improper to speak of "hypertrophy curves" in connection with these electrocardiograms. Likewise the designation of "right or left preponderance" is not accurate if this expression is associated with the idea that one ventricle is anatomically or functionally stronger than the other. At most the *potentials* in one or other of the ventricles predominate. But hypertrophy of one-half of the heart is not the sole factor responsible for this, since an alteration of the cardiac position may have the same effect. Accordingly it seems advisable to speak of a dextro- or levocardiogram or of right or left axis deviation.[1] In respect of the latter designations it must be remembered that the expression "axis deviation" refers to the electrical axis of the heart and not to the anatomical axis of this organ. Since these expressions are commonly misunderstood, we prefer dextro- or levocardiogram as suggested by Lewis. In order to avoid misunderstandings it might even be better for the beginner to speak of the "right or left type" of ventricular electrocardiogram.

The dextrocardiogram is usually found in association with lesions of the mitral valve. The responsible factor is not simply a hypertrophy of the right heart, but the clockwise rotation of the heart around its axis, associated with enlargement of the right ventricle, is likewise important; in an identical manner the levocardiogram seen in connection with insufficiency of the aortic valve or hypertension is not produced solely by hypertrophy of the left ventricle, but also by the counter-clockwise rotation resulting from enlargement of the left ventricle. However, since precisely the same dextro and levocardiograms (right and left axis deviation) are observed in healthy individuals, no diagnostic conclusions can be drawn from a tracing of this kind. They can scarcely be evaluated for the purpose of differential diagnosis.

The presence of a dextro- or levocardiogram does not prove the existence of a right- or left-sided cardiac hypertrophy; on the contrary massive hypertrophy of the right or left heart may exist without right or left axis deviation; this happens, for example, when the position of the heart tends to neutralise the electrocardio-

[1] Throughout this book the words "levocardiogram" and "dextrocardiogram" are employed merely as concise expressions to designate a high R wave in Lead I and deep S wave in Lead III and a deep S wave in Lead I and a high R wave in Lead III, respectively.

graphic alterations consequent to the hypertrophy, *accordingly the discovery of a "dextro or levocardiogram," of a "right or left axis deviation," does not have any important diagnostic value*

A low diaphragm tends to cause the picture of a dextrocardiogram, whereas a high diaphragm may cause a levocardiogram

Occasionally formulæ are employed to ascertain the degree of axis deviation, the authors are not convinced of their advantages in clinical diagnosis

In a simple dextro- or levocardiogram (Fig 34) the amplitude of the R wave in Lead II may be increased or diminished But if a distinct S wave is also *present in Lead II*, as in Fig. 36, this finding

a b

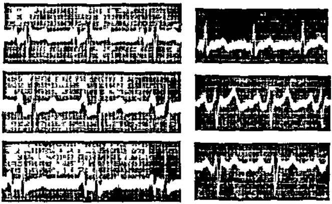

Fig 36a and b Right axis deviation is present in Fig 36a in a case of mitral stenosis left axis deviation is evident in Fig 36b a patient presenting a lesion of the aortic valves The P waves of Fig 36a are *very large (see p 295) U waves are present in Fig 36b*

has been said to give definite support to the idea that hypertrophy of the right or left ventricle is present rather than a variation of the normal electrocardiogram (Pardee) The positive finding alone may be utilized *with caution* Sometimes marked hypertrophy of the right or left side of the heart may exist without a deep S wave occurring in Lead II

During the early months of life, a dextrocardiogram is regularly noted *since for a time the right ventricle of the new born forms the* most massive part of the heart just as it did before birth Usually *six to nine months* elapse before the signs of the dextrocardiogram vanish As individuals become older the levocardiogram is encountered with increasing frequency although they may be healthy from the standpoint of circulation

Significance of Intraventricular Spread of Excitation

Apart from hypertrophy of one ventricle or from the position of the heart the structure of the ramifications of the conduction system and the manner of spread of the activation in the heart are important factors in regard to the size and form of the individual waves and thereby for the production of the levo or dextrocardiogram * Right or left axis deviation may appear from disturbances of conduction in the small branches of the transmitting system Rothberger and Winterberg by dividing the smaller ramifications of both main stems of the bundle branches were able to produce alterations in the amplitude of the individual waves of the initial complexes without other changes in the electrocardiogram These anomalies of conduction may be produced by a congenital abnormality of the anlage of the conduction system or by foci of disease in the conduction paths *Fig 37 illustrates an example of the transient appearance of a*

Fig 37 (Lead III) Alteration of the ventricular complex by a disturbance of intraventricular conduction

levocardiogram as the result of a disturbance of intraventricular conduction

The electrocardiogram was obtained from a case of tuberculous myocarditis at necropsy innumerable recent and old inflammatory foci were discovered in the finer ramifications of the conduction system in the ventricle Owing to the presence of these foci the electrocardiogram changed continuously and variations of the ventricular complexes were constantly observed even during complete rest The alterations were accentuated and occurred more frequently when the rate was increased by effort amyl nitrite, etc This case was described in detail by Grassberger

Sometimes a levocardiogram was present, but a few minutes later the electrocardiogram would be normal In Fig 37 there is a sudden transition from initial deflections with deep S waves to those with high R waves of normal shape (Lead III is reproduced) The cardiac rate and the conduction time remained unchanged An alteration of the functional capacity (irritability) of a branch of the intra

ventricular conduction system resulted in a momentary disturbance of conduction and transiently changed to a marked extent the appearance of the ventricular complexes. The tracing proves what importance the spread of the intraventricular excitation has in the appearance of the ventricular electrocardiogram. A change of the position of the diaphragm was not involved because the electrocardiograms were transcribed during calm respiration and either form of the ventricular complex appeared for minutes or hours.

If exceptionally a dextrocardiogram is found in conjunction with an insufficiency of the aortic valves or a levocardiogram in mitral stenosis, it is probable that disturbances of intraventricular conduction are responsible for these findings. Occasionally a dextrocardiogram is encountered in a case of syphilitic insufficiency of the aortic valves since myocardial lesions are common in this condition.

An electrocardiographic alteration produced by a disturbance of intraventricular conduction which during an animal experiment was induced by the destruction of a branch of the conduction system can be abolished by section of another branch (Rothberger and Winterberg).

Lastly two more factors have been considered as playing a part in producing dextro- or levocardiograms: first, a change in the relative positions of the two ventricles to one another resulting from the dilatation of the heart, and secondly the delay in the activation of the dilated ventricle consequent upon the lengthening of its path of conduction.

New Nomenclature

Earlier (p. 51) reference was made to some recent investigations especially by Wilson and confirmed by Storm, etc., in which it was suggested that the classical nomenclature of bundle branch block may be incorrect and that the experiments and clinico-pathological studies upon which the old nomenclature was based were incorrectly interpreted, and that tracings of the type exemplified by Fig. 22 or Fig. 23 should not be described as a block of the right bundle branch but rather of the left. According to Wilson the tracings which Lewis attributed to left ventricular effects and therefore called levocardiograms are actually due to the right ventricle and should therefore be called dextrocardiograms.

If a high R wave is found in Lead I and a deep S wave in Lead III this is said, according to the new nomenclature, to indicate an initial activation of the right ventricle, while a deep S wave in

Lead I and a high R wave in Lead III is said to characterize prematurity of excitation in the left ventricle

From these statements it would follow that according to the new nomenclature in cases of left ventricular hypertrophy we obtain a tracing which should be called a dextrocardiogram in cases of right ventricular hypertrophy (mitral valve lesions) a tracing is obtained which should be called a levocardiogram Some advocates of the new nomenclature explain this in the following way when hypertrophy of a ventricle has occurred its activation lags somewhat behind the other (in other words in hypertrophy of the left ventricle the right is activated somewhat earlier) Instances formerly diagnosed dextro or levocardiogram right or left axis deviation should be called right or left retardation Endeavours have been made in a variety of ways to advance proof that hypertrophy of one ventricle creates a situation in which the opposite ventricle controls the ventricular complex in the electrocardiogram and influences its appearance The lag of a ventricle which has become abnormal owing to hypertrophy and dilatation is ascribed for example to the fact that the conduction time in the bundle branch supplying the dilated ventricle is longer than in the normal bundle branch so that the affected ventricle is excited later than usual However since the dextro or levocardiogram is also encountered in the absence of dilatation and when hypertrophy alone is present other hypotheses must be employed Wilson and co workers believe that hypertrophy of one ventricle increases the mass of the septal wall of the opposite ventricle without increasing the bulk of the lateral walls thus increasing the magnitude of the electrical potentials of the non hypertrophied ventricle Up to the present time it has been just as impossible to obtain proof for the interpretation of curves of hypertrophy as in the case of the new nomenclature for bundle branch block tracings Here also unanswered objections still exist so that the writers retain the old nomenclature until decisive proof is submitted for the new

Further Changes in the Electrocardiogram in Marked Hypertrophy and Dilatation of One Ventricle

Earlier in the discussion it was pointed out that the electrocardiogram of hypertrophy of the right or left heart is not significant in itself because the dextro or levocardiogram may appear in the healthy individual Apart from this widening of the initial deflection and change in the S–T interval and the T wave are

absent But there are cases in which both appear that is the alteration of the form of the initial complex as well as of the terminal deflection

During the course of a considerable hypertrophy of the right or left ventricle the initial deflection may become broader and persist *as long as 0 10 second According to Pardee the initial deflection* may become even 0 11 second in width simply as the result of muscular hypertrophy of the left ventricle Since a duration of 0 10 second occasionally appears in a healthy individual it has significance only when it was absent in an earlier electrocardiogram of the same patient that might be available for comparison In this instance the widening may be explained by the fact that the activation requires a longer time in the hypertrophied thick ventricle *than in a normal muscle It is also assumed that the dilatation* often associated with hypertrophy produces a widening of the initial deflections since a disturbance of intraventricular conduction may result from overstretching of the smaller branches of the bundle which become incapable of conduction as the result of considerable dilatation of the ventricles Naturally, the widening of the initial complex is less marked in right ventricular hypertrophy for this is never as extreme as left sided hypertrophy

The S–T segment as well as the T wave may be altered in cases of cardiac hypertrophy *They show a tendency to proceed in a direction opposite to that of the initial deflection* Accordingly in a case of hypertrophy of the right side of the heart one may find the terminal deflections (S–T and T) above the zero line in Lead I and below the isoelectric line in Lead III (at times also in Lead II) in hypertrophy of the left heart depression and inversion of the terminal deflections in Lead I at times also in Lead II may be *present with an elevation in Lead III The participation of Lead II* depends upon the extent of alterations in Leads I and III

These alterations often begin in *that lead* which displays the high wave (that is in Lead I in the levocardiogram in Lead III in the dextrocardiogram) The S–T segment changes first and then the T wave The changes in the final deflection may be missed in the lead with deep S waves (Lead III in the levocardiogram Lead I in the dextrocardiogram)

Fig 38a was obtained from a patient who had a moderate mitral stenosis and considerable pulmonary stasis The rate is accelerated (100) and the P waves are unusually large and split There is a dextrocardiogram (deep S wave in *Lead I high R wave* in Lead III) The terminal deflection in Lead I is positive in

Lead III negative, the S–T segment is also depressed slightly below the zero line in Lead II

The alterations are much more distinct in Fig 38b, which was recorded in a patient suffering from chronic nephritis with hyper tension, massive hypertrophy and dilatation of the left ventricle. The initial deflections are 0 10 second in width and show the con figuration of a levocardiogram, that is, a high R wave in Lead I and a deep S wave in Lead III, the terminal deflections in Leads I and III are directed opposite to the initial deflections

The appearance of an abnormal terminal deflection in cardiac hypertrophy may be anticipated on the basis of our present know-ledge of the development of the normal electrocardiogram The T wave, like the initial deflection, develops from the algebraic sum

a b

FIG 38a and b The electrocardiogram of a right and left sided hyper trophy with oppositely directed terminal deflections

of the right and left ventricular T waves The preponderance of potentials in a hypertrophic ventricle must therefore act, not only on the initial but also on the terminal deflection

Certain authors (Barnes and Whitten) attribute these alterations of the terminal deflection simply to the augmented strain on one ventricle According to other observers the appearance of the alterations of the terminal deflection just described depends upon a myocardial damage because the affected patients succumb to their malady in a short time However, since these patients always have *advanced* hypertrophy and dilatation these observations do not prove very much On the contrary, it is not rare to encounter patients with marked hypertrophy and dilatation together with the above described alterations of the terminal deflection, although the cardiac status does not change for years, since it concerns

cases with valvular lesions or hypertension in which the myocardium would immediately fail, this survival would not be possible were the muscle badly damaged

Considerable difficulty is presented in the explanation of the question *why the opposite direction of the terminal deflection does* not occur in all cases exhibiting predominant hypertrophy of *one* ventricle The fact that this situation occurs in only some of the cases suggests that hypertrophy alone is not the cause However, clinical experience indicates that the degree of hypertrophy is responsible since the alteration is rarely encountered in mild hypertrophy but is associated much more commonly with marked hypertrophy. It is also stressed that the alterations are much more *frequent when the hypertrophy is associated with dilatation* This assertion seems to be correct, nevertheless it must be conceded *that at times oppositely directed terminal deflections are found in* lesions of the aortic or mitral valves in the absence of marked dilatation of the left or right ventricle The assumption of some investigators, namely, that when alterations of the after deflection are present a coronary sclerosis exists which has injured the con duction pathways, in many cases certainly does not apply Apart *from histological studies the inaccuracy of this assertion is attested* by the fact that oppositely directed terminal deflections are observed in youthful individuals suffering from congenital heart lesions and in mitral stenosis The observation that only a fraction of the cases of hypertrophy exhibit these alterations may be attributed to the fact that frequently there is also hypertrophy of the opposite ventricle which produces changes more or less neutralising the alterations ordinarily evoked by hypertrophy of only *one* ventricle The algebraic *sum of the two effects may approximate zero and a modification in* one direction may be cancelled by oppositely directed phenomena

In our opinion, disturbances of the intraventricular spread of activation play a very large *rôle* in the development of the oppositely directed *terminal deflection*

It can be demonstrated by tracings presenting rapid change in the form of the S–T segments and of the T waves owing to disease of the intraventricular conduction paths that disturbances of these *areas influence, not only* the initial deflection, but also the terminal excursions

Figs 39a and b were transcribed from the same patient in whom the tracing of Fig 37 was recorded As already mentioned, the form of the ventricular complex varied constantly For example, *a dextrocardiogram would at times change suddenly*

into a levocardiogram Fig 39a shows a levocardiogram with an initial deflection 0·10 second in width followed by an oppositely directed terminal deflection. Fig. 39b was recorded immediately

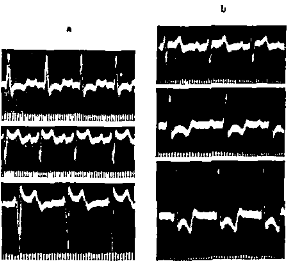

Fig 39a and b A disturbance of intraventricular conduction produces alternately right and left axis deviation with oppositely directed terminal deflections

afterwards from the same patient and shows a dextrocardiogram, again with an oppositely directed S-T segment and T-wave.

Fig. 40 was also obtained from the same patient; despite a

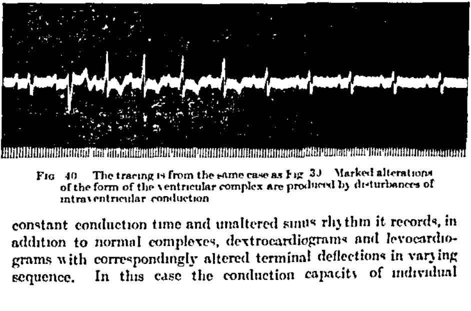

Fig 40 The tracing is from the same case as Fig 39 Marked alterations of the form of the ventricular complex are produced by disturbances of intraventricular conduction

constant conduction time and unaltered sinus rhythm it records, in addition to normal complexes, dextrocardiograms and levocardiograms with correspondingly altered terminal deflections in varying sequence. In this case the conduction capacity of individual

branches of the intraventricular conduction system at times changed so rapidly that ventricular complexes of many different forms directly follow each other. Tracings of this kind were frequently obtained from this patient.

This observation shows that not only the dextro and levocardiogram but also the oppositely directed terminal deflections may vary from beat to beat and that the type of intraventricular conduction (and not exclusively the hypertrophy and dilatation of one half of the heart) can produce the alterations of the terminal deflection described above. Whether or not an injury of the intraventricular conduction must be present in each case or whether additional factors can produce similar alterations cannot on the basis of our present knowledge be determined. It is conceivable that *marked dilatation of a ventricle may through stretching* produce atrophy of individual fibres of the conduction system which leads to disturbances of conduction of a kind that give rise to oppositely directed terminal deflections.

Since in a right or left sided hypertrophy the oppositely directed terminal deflections develop very gradually and always in a definite sequence they cannot be brought about by inflammatory or degenerative changes in the conduction system. Foci of such origin *would not develop so regularly at the same places and always cause* the same alterations. In other words the disturbances of intraventricular conduction in right or left sided hypertrophy and dilatation *must in some way be related to these conditions*.

An application of this conclusion may be made to patients with old fixed hypertension, chronic hypertensive nephritis or stenosis and regurgitation of the aortic valves, that is in conditions which impose a heavy burden on the left side of the heart. If a *levocardiogram* is found with *initial* deflections which are widened to 0.10 second and with terminal deflections in which the S-T segment and T wave is depressed below the isoelectric line in Lead I (at times also in Lead II) (Fig 38) while the terminal deflection is elevated above the zero line in Lead III these alterations may be regarded as the result of hypertrophy and dilatation of the left ventricle; an additional myocardial disease need not be postulated. The same situation prevails in stenosis of the *mitral valve and in pulmonary* emphysema. In these instances the corresponding alteration of the right heart alone can produce an elevation of the S-T segment and a positive T wave in Lead I and a depression of the S-T segment and an inverted T *wave in Lead III (at times even in Lead II)* (Fig 38).

But if these tracings which are not uncommon are found in patients in whom no reason exists or has existed for hypertrophy of the left or right heart (for example, a vanished hypertension) it is permissible to conclude from these electrocardiograms with abnormal terminal deflections the existence of myocardial damage

Periodically 'oxygen hunger' (anoxia) of the heart muscle or 'coronary insufficiency" have been assumed to be present because of abnormal terminal deflections in cases of cardiac hypertrophy This explanation also is not satisfactory because an oxygen deficiency of the myocardium maintained for years is scarcely conceivable without rapid failure or at least an increasing dilatation of the heart

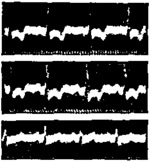

Fig 41 The electrocardiogram of a mitral aortic valve lesion without definite signs of structural injury of the myocardium except right and left sided hypertrophy

We believe therefore that no "second" disease need be added in order to account for the appearance of oppositely directed terminal deflections in hypertrophy of one part of the heart, these modified terminal deflections are the direct result of marked hypertrophy and dilatation When these alterations appear, the heart is always profoundly affected

Electrocardiograms of hypertrophy with oppositely directed terminal deflections are also shown in Figs 42 and 116

If both ventricles alike are altered by hypertrophy (for example, in a mitral aortic valve lesion) no typical modifications are found in the form of the terminal deflection they may be altered in the same direction (positive or negative) in all leads sometimes the main and final deflections are normal because of the summation of the oppositely directed effects of hypertrophy of both ventricles

Fig 41 was transcribed from a patient with a fully compensated mitral and aortic valve stenosis without signs of myocardial disease In this patient who presented hypertrophy of both ventricles the initial deflection is normal the S-T and T wave of Lead I and II are deeply negative in Lead III the terminal deflection is normal but in other cases it may also be altered

The finding of a right or left axis deviation without other abnormalities provides no information because it is frequently observed in healthy individuals If deep S waves are present, not only in Lead I or III but also in Lead II some probability may be attached

to the assumption of a hypertrophy providing no disturbance of intraventricular conduction is present Widening of the initial deflections to 0 10 second, and the finding in Leads I and III of terminal deflections directed opposite to the main deflection can be produced by a massive hypertrophy and dilatation of a part of the heart To be sure hypertrophy and dilatation of the right or left heart alone does not always produce such electrocardiograms but some associated though at present unknown factor (probably a disturbance of the spread of the intraventricular conduction) may be responsible, the presence of a second disease need not be postulated

THE DEEP Q-WAVE IN LEAD III

A small Q wave may be seen in any lead *of the normal electrocardiogram* But as it is often absent no deduction can be drawn from its failure to appear In hypertrophy of the left heart it is regularly found in Lead I, *and in right sided hypertrophy in Lead III* In bundle branch block it is also a common occurrence in the lead with the high R wave

In Lead III the Q wave may attain abnormal *depth and in this instance it may have diag*nostic significance According to the criteria of Pardee a Q wave in Lead III should be regarded as abnormal when (1) its depth exceeds 25 per *cent of the amplitude of the highest R wave* of the patient concerned (the R wave in any lead and not only of Lead III) (2) when it is the first downward deflection of the electrocardiogram *which is followed by a definite R wave* and (3) when there is no dextrocardiogram as often occurs in marked right ventricular hypertrophy (tricuspid lesions and congenital cardiac defects)

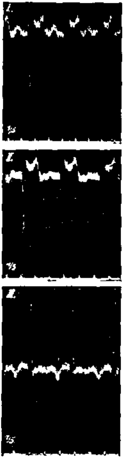

FIG 42 The electrocardiogram of a mitral tricuspid *valve lesion with* signs of right sided hypertrophy and a deep Q wave in Lead III The P wave is negative in Lead III and presumably inverted in Lead II

Fig 42 shows an electrocardiogram of a severe mitral tricuspid lesion confirmed by necropsy It reveals a right axis deviation with a deep S wave in Leads I and II and a high R wave in Lead III with oppositely directed terminal deflections (caused by right sided

cardiac hypertrophy) In this case the deep Q_3* has no significance

In the evaluation of a deep Q wave in Lead III, one must always suspect that an upward displacement of the diaphragm with a transverse position of the heart may be responsible for it It was stated earlier that a rotation of the heart around its axis can produce a dextro or levocardiogram a rotation of the heart around an antero posterior axis can also produce a deep Q wave in Lead III (Meek and Wilson), accordingly it is not rare to observe a Q_3 grow smaller or larger or appear and vanish with respiration To be sure a pathological Q_3 can also become larger or smaller during respiration

Fig 43 shows Lead III of a healthy man fifty six years of age, whose circulation was normal from a clinical standpoint The electrocardiogram was transcribed during quiet, normal respiration At the beginning of the tracing (expiration) an R wave is seen,

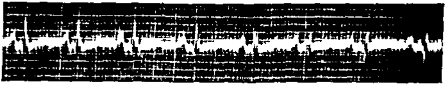

Fig 43 (Lead III) The appearance of a deep Q wave during inspiration

during inspiration it becomes smaller, simultaneously a distinct Q wave develops

Not rarely one finds a deep Q_3 in a healthy woman during the last months of pregnancy as the result of upward displacement of the diaphragm and the transverse position of the heart

Deep Q waves are a common and normal finding in young children even as late as the sixth or seventh year of life (Krumbhaar and Jenks)

If all these reservations are considered then a deep Q_3 is a very valuable finding from a diagnostic standpoint In a vast majority of cases in adults coronary sclerosis is present According to Willius a deep Q_3 constituted the sole abnormal electrocardiographic sign in 198 out of 268 cases of hypertension and angina pectoris Only three patients among 300 with a deep Q_3 had normal hearts Since the appearance of a deep Q_3 may be the result of considerable elevation of the diaphragm and since it may appear and vanish with respiratory movements caution must be exercised in the evaluation of an otherwise normal electrocardiogram which shows *only* a deep Q wave even if it fulfils the requirements of Pardee Never

* In this form of expression the index figure is intended to denote the lead under discussion Thus Q_3 represents the Q wave in Lead III

theless, the discovery of a deep Q_3 will always impel one to be careful and awaken the suspicion of a myocardial disease, this is particularly true if anginal complaints, hypertension, or diabetes is present If still other alterations, though slight, are present in the electrocardiogram, a deep Q_3 may decisively favour the diagnosis of a myocardial disease

Fig 44 shows short sections of tracings from patients with an abnormal Q_3 The upper series reproduces tracings which show a

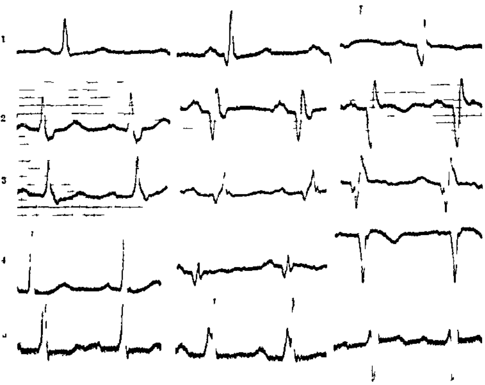

FIG 44 The electrocardiograms of five cases with abnormal Q waves in Lead III The leads of each case are placed alongside of each other

deep Q_3 (also a distinct Q_2) without other alterations in the electrocardiogram The T waves in Leads I and II are rather low The notching of the descending limb of the R wave near the base line is not abnormal The tracing was recorded in a man of sixty four who four years earlier had suffered from coronary thrombosis from which he had completely recovered Since that time he noted only after great exertion pain which radiated into the left arm

The second series shows a deep and broad Q wave in Leads II and III in addition to other changes in the electrocardiogram The

width of the initial deflections amounts to 0.12 second and T_2 is practically absent. In this case (an advanced coronary sclerosis, old coronary thrombosis?) the presence of a severe myocardial disease would have been assumed even in the absence of a typical Q_3.

The deep Q wave in Lead III, like any other wave of the electrocardiogram, may at times be notched and widened. This is illustrated by the third series of tracings in which the initial deflections are 0.12 second in width; moreover, the auriculo-ventricular conduction time is prolonged to 0.22 second and the T wave in Lead II is very low.

In the fourth series it is difficult to determine whether or not a

A B

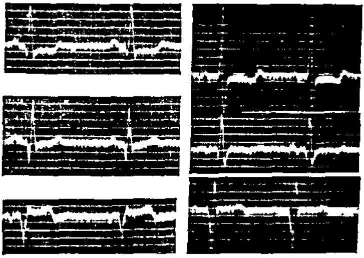

FIG. 4 a and b. Abnormal electrocardiograms with deep Q_3

deep Q wave exists in Lead III. Although in this lead a deep downward deflection may be readily seen, no distinct R wave precedes it (which would favour an S wave) but likewise no distinct R wave follows it (which would favour a Q wave). Since a distinct Q wave may be seen in Lead II, the deep negative wave in Lead III may well represent a Q wave. Moreover, the T wave in Lead II is abnormal (slightly inverted). The patient from whom the tracing was transcribed suffered from coronary sclerosis.

The fifth series was obtained from a patient with a marked stenosis of the aortic valves and a coarctation of the aorta. In Lead III a short upright wave may be noted; this, according to the rule, should be called an R wave; a downward wave follows it and then a very high positive one occurs. Tracings of this kind are not

uncommon In our experience the higher wave, that is the second upright wave, must in this case be regarded as an R wave Accordingly a deep Q_3 is present, then the first upright deflection would be an "innominate" wave since it has not received a special name In other words a positive wave may precede a Q wave only when it is followed by a much larger positive one in which circumstances the latter is the R wave (see Fig 28) Small upright waves of this kind regularly precede the deep Q wave in the third lead of right bundle branch block tracings recently described by Wilson (see p 58)

Moreover in this series of tracings (Fig 44 series 5) the conduction time is prolonged to 0 23 second and the terminal deflection is depressed below the zero line in Lead I and is elevated in Lead III

In Fig 45a (the tracings at the left) the diagnosis of a deep Q wave cannot be made from Lead III since, while the first wave of the initial deflection is directed downward no distinct R wave follows it A distinct Q wave is evident in Lead II and is just visible in Lead I Accordingly the existence of a deep Q wave may also be assumed in Lead III The S–T segment is depressed in Lead I and elevated in Lead III

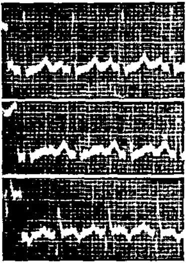

FIG 46 B C I T_2 and deep Q_3 in a child with rheumatic heart disease

In Fig 45b (the tracings on the right) a deep Q wave is undoubtedly present *in Lead III but a deep* S wave is apparent in Lead II and *is also prominent in Lead I Here also* the S–T segment is below the isoelectric line in Leads I and II T_1 and T_2 are diphasic

Deep Q_3 waves frequently appear in children with myocardial involvement in diphtheria or rheumatism Fig 46 is a reproduction of a tracing transcribed from a child seven years of age suffering from rheumatic heart disease in an acute stage The T wave in Lead II is low and bifid the Q wave in Lead III is abnormally deep But since a deep Q wave in Lead III and a bifid T wave in Lead II may be found in normal children the diagnosis of a myocardial lesion is impossible

There has been considerable discussion concerning the possibility of the localization of the myocardial disease which leads to a deep

Q wave The view that a disease of the posterior part of the ventricular septum causes the appearance of a pathological Q wave has found most support Other investigators adopt a different explanation Wilson believes that the order or speed with which the inner surface of the free wall of the left or right ventricle is activated determines whether a Q wave will appear in Lead I or III Moreover, it is known that not only coronary sclerosis as is often asserted but other types of myocardial disease can also produce a deep Q_3

The significance of the Q wave in the electrocardiogram of coronary thrombosis will be mentioned later (p 128)

WIDENING AND SPLINTERING OF THE INITIAL DEFLECTION (DISTURBANCES OF INTRAVENTRICULAR CONDUCTION)

As long as the conduction system is intact so that the excitation may reach all parts of the ventricle rapidly and undisturbed the initial deflection of the electrocardiogram is recorded in thin delicate lines Alterations of cardiac position and extracardiac factors simply modify the amplitude of the individual waves causing one to become low or to vanish another to enlarge, but their form does not change nor does the initial deflection materially widen the limbs of the wave are neither split nor thickened But if in the course of a myocardial disease pathological foci develop in the specific tissues disturbances of intraventricular spread of excitation result which produce initial deflections that are abnormal in width and shape

As emphasized elsewhere bundle branch block is a severe disturbance of intraventricular conduction in which the direct excitation is barred from one ventricle so that the stimulus at first spreads only in the one bundle branch whose conduction is intact

However pathological foci in the finer ramifications of the atrioventricular system also lead to disturbances of intraventricular conduction giving rise to widening and splintering of the initial deflections

If a pathological focus has interrupted the conduction of excitation in a small branch of a bundle a small area of muscle will not be excited directly through the conduction system but only by the detour over the neighbouring muscle The potentials appear belatedly in the area of muscle whose activation is thus delayed and lead to a change of the normal spread of the electrical forces causing a widening notching and splitting of the normally slender and delicate waves of the initial deflections

However a *slight* widening and slurring of the initial deflections

can be designated as pathological only when an earlier electrocardiogram of the case has been recorded and these alterations were not then present If no such means of comparison are available, no conclusion can be drawn from slight widening and notching As stated on p 23, slight notching of the initial deflections near the base line may occur normally This is explained by *anomalies in the structure* of the specific tissue Likewise slight widening perhaps to 0 08 second should not be taken seriously in the absence of comparative tracings since this remains within the domain of the normal and possesses significance only when previously absent

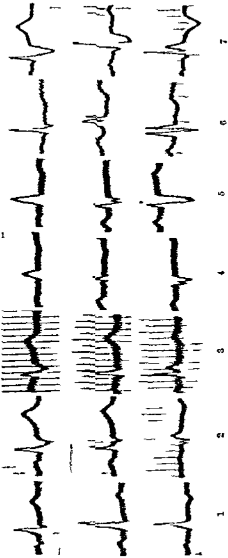

Fig 47 The electrocardiograms of seven cases of widening plumpness thickening and splintering of the initial deflection (the three leads under each other)

But if additional new foci appear in one or other of the ventricles the widening as well as splitting notching and thickening of the initial deflections acquire a degree which no longer *occurs in the normal tracing* and immediately permits the diagnosis of a myocardial disease *These alterations never appear without myocardial disease or without foci in the ramifications of the atrioventricular system* (The only exception to this rule a congenital *anomaly, is discussed on p 325*) Here again the electrocardiogram alone does not indicate the type of foci present whether an old, healed or a recent progressive process exists The electrocardiogram makes possible the localization of a disease but it does not provide any information as to the nature or the prognosis

The initial deflection may be more than 0 12 second in width Very often a right or left axis deviation is present but both may be absent Splitting or notching may be very obvious or may not be evident Usually but by no means invariably changes are also present in the terminal deflection

Fig 47 shows sections of seven tracings with abnormal initial deflections In each instance the three leads have been placed below each other

In Case 1 the initial deflections are somewhat plump Their width is not increased (0 08 second) nor are they split or notched The limbs however are slightly thickened and differ distinctly from the normal initial deflection of Fig 5 Likewise T_2 is low Certainly the changes are slight and the electrocardiogram is borderline Even if the initial deflections are only 0 08 second wide, they appear plump if only *one* wave (an R wave) is present and it is not split into several deflections

In Case 2 the initial deflection is 0 11 second in width In Leads I and II a notched plump S wave in Lead III a short split Q wave is visible The terminal deflection is normal The electrocardiogram in this case is unequivocally abnormal Formerly the small wave following the R was erroneously called a negative P wave The electrocardiogram recalls that form of right bundle branch block which Wilson has recently described (Fig 28)

The electrocardiogram in Case 3 is also abnormal Here the initial deflection is 0 12 second in width and is notched in Leads I and III In Lead II the QRS complex appears normal since the plump broad S wave of Lead I is removed by summation with the thickened wave in the descending limb of the R in Lead III Here also the terminal deflections are normal (Wilson s block)

In Cases 4 and 5 the plumpness thickening and splitting of the initial deflection is easily recognized In Case 4 the conduction time is prolonged to 0 22 second The terminal deflections are abnormal the S–T segment in Leads I and II being displaced below the zero line and the T waves being absent in both leads

Similar alterations are evident in Case 7 where in Leads II and III the initial deflection exhibits splitting producing an M form in contrast to the W form of Cases 4 and 5 (Leads III or II) In Case 7 the initial deflection is 0 14 second in width The S–T segment and the T wave are normal in Leads I and II (Wilson s right bundle branch block)

In Fig 48 the heart rate is accelerated The initial deflection is 0 14 second in width and is split in all lead Moreover, it is directed

mainly downward (S waves) in all leads The terminal deflection shows an elevated S–T segment in Leads I and II

Only the *positive* finding of widening and splintering of the initial deflection is decisive for the diagnosis of morbid foci in the heart muscle On the other hand it is not permissible to conclude that the heart muscle is intact if no alterations are present in the initial deflection *Even the most extreme myocardial disease may be accompanied by initial deflections of normal width and shape* If the experiment of Eppinger and Rothberger is repeated and profound injury of the myocardium is produced by the injection of corrosive substances into large areas of both ventricles the initial deflection is altered to the extent that some of the waves become larger or smaller etc But widening or thickening of the initial complex does not appear since the excitation by means of the normal conduction system proceeds with normal velocity to all those parts of the ventricle still capable of response The slight alterations in the amplitude of the waves are produced simply by the disappearance of potentials of the necrotized sections of heart muscle

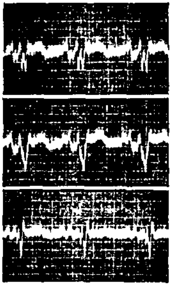

FIG 48 Disturbance of intraventricular conduction (myocarditis)

Only when pathological foci occur in the specific tissue do alterations (splintering and widening) appear in the initial deflections Since the atrioventricular system is separated from the common muscle and since it has its own blood supply it does not necessarily become involved in diseases profoundly affecting the common muscle Accordingly cases of coronary sclerosis may be observed in which the cardiac muscle is very necrotic and so full of scars and cicatrices that scarcely a single healthy area can be found and yet—for the reasons mentioned—during life the initial deflections were not widened In other cases a very considerable widening of the initial deflections may appear without any essential alteration of the common muscle This may occur if the disease exclusively involves the specific tissues as for example, when the vessels of the conduction system *alone* are affected

When in a levocardiogram the widening and splintering of the initial deflection gradually increases and the terminal deflection

is correspondingly changed, a picture almost of the common type of bundle branch block is reproduced Fig 47 (Case 5) shows a transitional form The difficulties of differential diagnosis were stressed earlier (p 58) *Often a distinction is impossible* Earlier the point was stressed not to make the diagnosis of bundle branch block too frequently and not to diagnose "incomplete bundle branch block" in every patient with a widened or a split initial complex

ABNORMAL ELECTROCARDIOGRAMS WITH SMALL EXCURSIONS

Elsewhere (p 47) reference was made to the fact that severe myocardial disease may be associated with small deflections in the electrocardiogram Before an attempt is made to interpret this finding, a careful standardization of the apparatus (with the patient included in the circuit) is essential Myxoedematous alterations of the skin or the presence of a pericardial effusion must be ruled out The excursions must be very small in *all* leads Small deflections in only one lead may be produced by a particular position of the heart and as mentioned earlier, also occur in healthy individuals

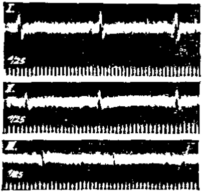

Fig 49 Small excursions in a case of myocardial disease

Fig 49 shows very small excursions in a case of coronary sclerosis The T waves are almost invisible and the initial deflections are very small

Also in Fig 47 (Case 4), apart from other signs of myocardial disease, the deflections are small

Small deflections of the type mentioned are regarded by some writers as a consequence of a diffuse disease of the myocardium for example, myocardial fibrosis While this interpretation is sometimes valid, for many cases it certainly does not hold good In coronary thrombosis, that is, when the myocardial damage is circumscribed, the excursions frequently are small If such a patient recovers, the amplitude of the deflections gradually becomes greater, an occurrence which would be impossible in a myocardial fibrosis If a patient whose electrocardiogram after a coronary thrombosis had shown small excursions dies, occasionally one finds at necropsy

a *single* large but circumscribed necrosis together with a few cicatrices but not diffuse myocardial injury The reason for the development of 'small deflections in the electrocardiogram of myocardial disease is still unexplained

ARBORIZATION BLOCK ELECTROCARDIOGRAMS

In 1917 Oppenheimer and Rothschild described electrocardiograms which showed initial deflections which were markedly split very wide and low These alterations were ascribed to a disease not of the main bundle branch but of the finer ramifications of the atrioventricular system and for this reason it was called arborization block This explanation was contested for a long time and the specific classification of these tracings was frequently denied even at present many authorities consider that an arborization block should never be diagnosed as a separate electrocardiographic entity Repeatedly tracings of this kind have been erroneously ascribed to partial bundle branch block In the authors view recent investigations have tended to confirm the opinion of Oppenheimer and Rothschild

In animal experiments an arborization block can be produced only with great difficulty even very extensive lesions of the ramifications of a bundle branch never evoke it This observation alone proves that more than a single partial bundle branch block exists in arborization block cases In animal experiments a wide variety of disturbances of conduction in the ramifications of a bundle branch are readily produced but the electrocardiographic picture described by Oppenheimer and Rothschild is never found However suitable experimental arrangements show that arborization block electrocardiograms appear if a *very extensive* disturbance of the spread of excitation occurs in the periphery of *both* ventricles for example they are produced when one branch is completely interrupted and only a small branch of the other remains intact and constitutes the sole path of conduction of auricular stimuli to the ventricle (Scherf Mahaim) In this instance from the solitary circumscribed area at which the impulse reaches the ventricle it can spread over the ventricles only *via* the common muscle this might very well lead to the tracings described (Rothberger) since the conduction of the common muscle is much slower than that of specific tissue

It is understandable that such extensive injury of both ventricles can appear only in severe myocardial diseases Actually the arborization block electrocardiogram is encountered only in severe coronary

sclerosis, in luetic myocardial disease, in most severe myocarditis Only rarely does it appear as a result of earlier disease, *e.g.* diphtheria, usually a progressive malady is present Despite the absence of other signs of serious heart disease, the discovery of an arborization block compels one to offer a poor prognosis

In diffuse injury of numerous branches of the A-V system, large areas of muscle are no longer simultaneously activated, for this reason no large potentials are formed and the deflections are small This occurs because the activation of single areas of both ventricles proceeds sequentially rather than simultaneously, and the excitation cannot proceed along the rapidly conducting specific fibres, such markedly abnormal spread of excitation leads to notched split and thick waves

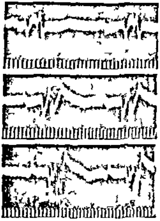

Fig 50 Arborization block

In Fig 50 there is an accelerated sinus rhythm with abnormal P waves, the auriculo ventricular conduction time is prolonged to 0 26 second The initial deflections are low, 0 17 second in width, notched thickened, and split The S-T segment in Lead I is depressed below the zero line and the T wave is inverted

Accordingly arborization block presents (1) very broad and (2) very small initial deflections The width of the initial deflections always exceeds 0 12 second and may attain 0 20 second, whilst the amplitude should not exceed 4 mm (*measured in the upright excursions from the upper border of the isoelectric line, and in the inverted waves from the lower border*) The S-T segment like the T wave, may be entirely normal but both may exhibit changes A levocardiogram or dextrocardiogram may, but need not be present

Since the limit of 4 mm in amplitude is somewhat arbitrary, obviously the decision whether or not a bundle branch block with low initial deflections or an arborization block exists is, at times very difficult All transitions between the various forms occur Since the minimum width of 0 12 second also is arbitrary, transitions also exist between simple widening of the initial deflections, considered in the preceding sections, and arborization block

In Figs 51 and 52 additional tracings of arborization block are

reproduced Here also the marked widening and splitting of the very low initial deflections is striking The tracings show that the terminal deflection need not as in bundle branch block be directed opposite to the initial deflection But when the excursions are somewhat larger and a right or left axis deviation with oppositely directed terminal deflections is present it may at times be difficult to determine whether or not a bundle branch block exists

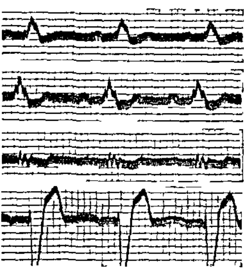

FIG 51 Arborization block (?) The lowest tracing is a chest lead

In Fig 51 a chest lead is included (see p 150) It is interesting to note the same large excursions that are seen in bundle branch block tracings We have observed this finding frequently in arborization block tracings It must be admitted that to some extent it stands in contradiction to the explanation of arborization block advanced in the preceding sections

It is quite possible that cases in which large deflections are found with chest leads (as in Fig 51) some disturbance of intraventricular conduction other than arborization block is present a bundle branch block with small deflections might be responsible We would suggest that the diagnosis of an arborization block be made only in cases where the chest lead *also shows the low* and widened ventricular complexes

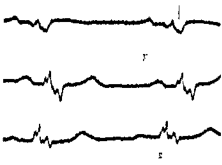

FIG 5 Arborization block

The *practical significance* of the presence of arborization block is great since it indicates very extensive and very severe myocardial disease In this point *consists the great difference between arborization and bundle* branch block since the latter may be caused by a single small lesion

THE ABNORMAL S-T INTERVAL AND THE ABNORMAL T-WAVE

General Remarks

In the earlier sections emphasis has repeatedly been placed upon the fact that diseases of the propulsive cardiac muscle produce no noteworthy widening or splitting of the initial deflections even if they cause necrosis of large areas of muscle *Only* when branches of the conduction system are involved, that is when disturbances of the intraventricular spread of excitation occur, are abnormally widened and split initial deflections found

It is still impossible to explain why no widening of the initial deflections appear following a general injury of the muscle alone (for example in fatty or brown degeneration) This behaviour could be understood if the affected area of muscle died, then it would be devoid of reaction for only those parts of the muscle which remain alive produce electrical forces, the initial deflections which develop differ from the normal but widening is absent The absence of widening of the initial deflection is incomprehensible however in cases in which the muscle is merely damaged but still reacting Experiments on excised strips of ventricular muscle show that intoxications for example with digitalis lead to the same disturbances of conduction as those which are found in the specific tissue On the other hand, it should not be forgotten that activation of the ventricle proceeds from within outward and the path of excitation in the common muscle is short

On the basis of these observations a normal status of the heart muscle cannot be deduced from the absence of widening or of splitting (notching thickening) of the initial deflections

However another section of the electrocardiogram is also associated with ventricular activity the terminal deflection This reacts more sensitively to alterations in the heart muscle especially to changes in the body of the muscle, and shows characteristic modifications of form more obviously than the initial deflection

Formerly the T wave alone was regarded as the ' terminal deflection But clinical experience during the last ten years has shown that the form of the S-T interval *the S-T segment* which lies between the initial deflection and the T-wave sometimes possesses greater significance in the appraisal of the heart muscle than the T wave itself Since the S-T segment gradually merges with the T wave and since under pathological conditions, the separation is often impossible for example, in bundle branch block, the two

sections are best considered together and discussed as the terminal deflection

The great sensitivity of the terminal deflection may be presumed *a priori* The initial deflection develops as the result of the activation process This proceeds with remarkable rapidity and indicates physiologically an anoxybiotic splitting of the available material This event is less easily disturbed than the more complicated and *more slowly evolving process of restoring material for the next excitation it is this latter process which produces or accompanies the terminal deflection*

In agreement with this conception it has since Samojloff s studies repeatedly been found that the most diverse interferences which fail to affect the initial deflection markedly change the form of the T wave To be sure under such circumstances an exact study reveals that the initial deflections show slight alterations in the form and size of the waves But these changes are less marked in the QRS complex that is completed in a short time and are less striking to the eye than the modifications of the sluggish terminal deflection Often the tracing must be carefully inspected in order to discover that one wave has become somewhat smaller another higher a small Q wave appears an S wave vanished etc There are no distinctly perceptible alterations of the direction of the excursion and the width *of the initial deflection*

It is easy to prove the independence of the terminal deflection If in an animal experiment certain branches of the sympathetic nervous system are stimulated very high T waves appear clinically large T waves are seen in hyperthyroidism and in many cases of cardiac neurosis with high sympathetic tonus If the branches of the sympathetic system to the heart of a dog are cut the T waves become deeply inverted If certain areas of the heart muscle are cooled or warmed the T waves become positive or negative cooling or warming other sections produces T waves with exactly the opposite form In the same way transient alterations of the T waves have been observed in men after ingestion of cold drinks since the cooled esophagus and *the cooled gastric wall can chill the adjacent sections of the heart muscle and thus alter the activation process (Wilson and Finch)* Digitalis like other poisons (for example morphine quinine) can decisively change the form of the terminal deflection In all of these instances no noticeable modification of the form of the initial deflection is observed

In spite of its great independence the form of the T wave depends to a vast extent upon the form of the QRS complex The

process of recovery from excitation which is responsible for the T wave is influenced by the course of the activation process itself and the form of the initial complex co-determines the form of the terminal deflection But this relationship should not be interpreted to mean that the appearance of the terminal deflection can be deduced from the form of the initial complex since in addition the form of the T wave is influenced by many other factors In the healthy muscle recovery from excitation proceeds differently than the excitation process itself (p 15) this difference is even more marked under pathological conditions Thus no predictable terminal deflection must invariably follow a certain type of initial complex Very often an abnormal terminal deflection is noticed after a widened and otherwise abnormal initial complex but as mentioned earlier the initial complex of arborization block the widest and most splintered known may be followed by an entirely normal terminal deflection There is no invariable connection between the amplitude of the initial deflection and the size of the T wave since a very large T wave *may* follow a very small initial deflection or a small T wave may occur after a large initial complex

Apart from the manner of spread of excitation in the heart and apart from the tonus of the cardiac nerves (Einthoven Rothberger and Winterberg) the form of the T wave is influenced by

1 The position of the heart A transverse position of the heart may produce an inverted T wave in Lead III a low diaphragm with a median placed heart may be associated with a very low T wave in Lead I (Fig 17) At times the inverted T wave in Lead III is observed only during a definite phase of respiration and corresponding to a certain position of the diaphragm (Fig 3?) The appearance of T_3 is also affected by alterations in the form of T_1 and T_2

2 The condition of the skin Not only in the completely developed form of myxœdema but also in cardiac patients with chronic decompensation the skin is dry and scaly so that the T wave may become very low (see p 46)

3 Effusions Effusions in the thorax œdema of the skin and especially pericardial effusions produce small and often abnormally formed T waves as well as a reduced amplitude of all the other waves

4 Cardiac hypertrophy and dilatation Earlier (p 73) reference was made to the fact that cardiac hypertrophy and dilatation may also produce alterations of the terminal deflection since the latter assume a direction opposite to the initial complex

Accordingly the finding in a case of left-sided hypertrophy of a negative terminal deflection in Lead I (and perhaps also in Lead II)

or in a case of right sided hypertrophy of a negative terminal deflection in Lead III (perhaps also in Lead II) does not without further evidence warrant the conclusion that myocardial disease (myocarditis myocardial degeneration etc) is present But if these alterations of the terminal deflection are found without any evidence of hypertension past or present without a lesion of the aortic valves or without any other known reason for the presence of cardiac hypertrophy they are always indicative of a myocardial disturbance (see p 78)

5 In the appraisal of a terminal deflection consideration must always be given to the possibility of a digitalis effect since digitalis can as will be shown (p 101) decidedly influence the terminal deflection

The Different Types of Abnormal S T Intervals and Abnormal T-waves

The *length* of the S T segment in the normal individual shows such decided variations that with few exceptions (see p 108) up to the present time it has been impossible to draw any conclusion from it The length increases with more prolonged duration of systole Any disturbance which shortens systole also shortens the S–T segment (tachycardia excitation of the sympathetics)

Since normally the S–T segment may lie slightly above or below the zero line caution must be exerted in the appraisal of its form before definite conclusions are drawn

The abnormal S T segment may lie above or below the zero line beyond the limits described on p 23 There are three possibilities —

1 The S T segment may depart from the descending limb of the R wave or the ascending limb of the S wave thereby showing a displacement in the same direction as the preceding main wave of the initial deflection and originating from it before it reaches the isoelectric line These changes are discussed in the chapter on coronary thrombosis pulmonary embolism and pericarditis

2 The S–T segment begins at the isoelectric level but it abruptly ascends immediately after leaving the R wave and forms an arch *whose ends are at the zero line but whose centre is displaced upward* in the same direction as the R wave This variety of S T segment is frequently found in pericarditis (see p 132)

3 The S–T segment may be displaced in a direction opposite to the main wave of the initial complex in other words with a positive *initial deflection it may run below the isoelectric line* whilst it may run above the line when the initial complex is directed downward

Nevertheless it begins at the isoelectric line These alterations of the S T segment are rarely the same in all leads and frequently but not invariably show an opposite direction in Lead I and III they are encountered in myocardial injuries of the most diverse types but *especially in diseases of large areas of the heart muscle* (diffuse coronary sclerosis extensive myocarditis brown atrophy in advanced carcinomatosis and general intoxications) If the myocardial disease is limited to *one* ventricle then the S–T segment is usually opposite to the main deflection in Lead I or only in Lead III In injuries which affect the *entire* heart frequently we find the same displacement of the S–T segments in *all* leads and consequently (according to the rule of Einthoven) most marked in Lead II

An abnormal S T segment may be followed by a normal T wave

Until recently these abnormalities of the S T segment were never mentioned they are now emphasized as common and important findings often they appear earlier than changes in the T waves and their incidence is higher Isolated changes of the T waves with normal S–T segments occur usually but not exclusively with circumscribed anatomical lesions (myomalacia myocarditis)

The abnormal T waves can be subdivided into five groups —

1 The abnormally low T wave (under 1 5 mm)

2 The complete absence of the T wave

3 The diphasic T wave (at first a negative and then a positive phase or less often the opposite)

4 The inverted T wave

5 The high positive T wave

Since normally or as the result of changes in the position of the diaphragm atypical T waves may temporarily be found in Lead III *only* Leads I and II come under consideration in the clinical evaluation

Since the T wave like the QRS complex is regarded as the sum of single potentials from numerous segments of muscle of both ventricles the disappearance of potentials as a result of disease of one or another area of muscle may cause a positive T wave to become even higher or a negative T wave to become positive Thus occasionally one sees very high positive T waves appear after a coronary thrombosis After an exercise test in patients with coronary stenosis the inverted T waves usually become high for a few minutes (see p 145) However since comparative values are not available as a routine and since a positive T wave represents a normal finding conclusions can hardly ever be drawn from a wave

Only the four other forms of abnormal T waves can be 1

The Abnormal Terminal Deflection in Different Lesions of the Myocardium

In Fig 53 short sections of tracings (with the three leads shown under each other) are reproduced from seven cases in which the T wave or the S–T segment are abnormal

In Case 1 a deeply inverted T wave appears in Leads II and III in addition to an abnormal Q wave in the same leads The initial deflection and the S–T segment is otherwise normal The patient had coronary sclerosis and suffered from anginal pain upon exertion No positive evidence of coronary thrombosis was present

In Case 2 there is an abnormal notch in the descending limb of the R wave in Lead I a short but broad and split Q wave in Lead III The S–T segment is slightly depressed in Leads I and *II* the T wave is almost invisible in Lead *I* and it is deeply inverted in *Leads II and III* The electrocardiogram was *obtained from a case of* acute myocarditis

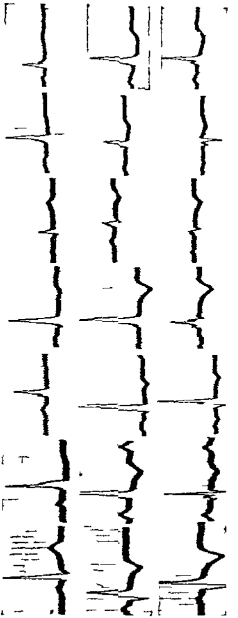

Fig 53 The electrocardiograms of seven cases with abnormal terminal deflections (the three leads are placed below one another)

In Case 3 a moderately severe stenosis of the aortic valve was present in addition to an acute myocarditis after tonsillitis The initial deflection is normal except for a small notch in the ascending

limb of the R wave in Leads I and III The S–T segment is normal in all leads In Lead I the T wave is just visible as a negative wave and it is inverted in Leads II and III When the myocarditis subsided the T wave became distinctly positive in Lead I barely so in Lead II

In Case 4 a mitral aortic valve lesion with decompensation was present The initial deflection is plump notched and 0 10 second in width The T waves are negative in all leads Since a severe hypertrophy of *both* sides of the heart existed the existence of a myocardial disease cannot without further information be diagnosed from the accompanying electrocardiograms (see Fig 41)

Case 5 had severe rheumatic fever Clinical examination of the heart disclosed nothing abnormal In the electrocardiogram there is a plump low initial deflection in all leads as well as a depressed S T segment and a negative T wave in Leads II and III This finding is indicative of a myocarditis

To determine whether the S T segment is located on above or below the zero line it is best for the beginner to observe the isoelectric line in diastole i e before the P wave of the following beat and to project this level *backward* to the S–T segment under investigation

Case 6 was a twenty four year-old girl with a decompensated hypertension caused by chronic nephritis Moreover a severe myocardial injury of unknown genesis existed The electrocardiogram was transcribed prior to the administration of digitalis to which the heart did not react The S–T segments are depressed in Leads I and II and slightly elevated in Lead III the T waves are almost invisible in Leads I and II and slightly inverted in Lead III The electrocardiogram does not in any way represent the type in which the after deflection is oppositely directed as in hypertrophy of the left heart but unequivocally reveals myocardial disease

Tracing 7 was obtained from a sixty five year-old patient who was suffering from extreme cachexia as a result of an inoperable gastric carcinoma The slight notch of the initial deflection is common at this age The depression of the S–T segments and the low T waves in all leads are produced by brown atrophy of the heart muscle this interpretation was confirmed by necropsy One part of the T wave is *probably* displaced below the isoelectric line with the S–T segment For this reason one can in such cases also speak of a diphasic T wave

If during the development of a myocarditis an opportunity is afforded of recording electrocardiograms repeatedly one can at times

observe the T wave gradually become lower then disappear and finally become inverted In recovery from a myocardial injury the *change in the opposite direction may be seen much more frequently* These changes do not necessarily follow one another in regular *sequence a positive T wave can become inverted without an* intermediate stage and *vice versâ* or more accurately the inter*mediate phase sometimes passes so rapidly that it cannot be demon*strated

*Some observers support the view that from a prognostic stand*point inverted T waves are less favourable than almost invisible T *waves since they indicate more profound myocardial damage* Serial examinations conducted in one and the same case may *demonstrate that this assertion is true But when the patient is* seen for the first time a very low as well as an invisible diphasic *or inverted T wave must be designated as abnormal but it is* unjustifiable to draw more far reaching conclusions from the various *forms*

In old age (in patients over sixty five) alterations in the terminal *deflection are frequently found more rarely also in the initial* deflection although the patient does not complain of cardiac *symptoms The alterations in the electrocardiogram are nearly* always attributable to coronary sclerosis they may remain un*changed for years and for this reason from a prognostic standpoint* should not be considered absolutely unfavourable They have more *serious significance only* when they develop and progress rapidly

Occasionally bifid and reduplicated T waves may be observed in the *course of* cardiac diseases (Fig 80) They are found in normal children Fig 80 (Case 5) was obtained from a three year old child ill with acute rheumatic fever right sided lobar pneumonia and empyema In addition to a sinus tachycardia one may perceive a reduplication of the T wave in Lead II and in the chest leads taken from the cardiac apex and the left sternal border

One cannot draw conclusions concerning the type of cardiac disease present either from the disturbance of intraventricular conduction or from abnormal T waves that is from the electrocardiogram alone Abnormal T waves are found in general diseases of the heart muscle (in the sense of a degeneration or intoxication) as well as in localized processes (myocarditis myomalacia diphtheritic necroses etc) An abnormal S T segment is found in generalized damage of the entire heart or of one chamber more rarely in local processes and only during definite stages of the disease

If in a patient with an otherwise unexplained persistent tem

perature an abnormal T wave appears, a myocarditis may be assumed even in the absence of other cardiac findings. Abnormal T waves occurring in patients with angina pectoris suggest an organic alteration of the cardiac muscle consequent to coronary sclerosis or to lues. If a patient consults us for gastro intestinal complaints such as a feeling of fullness and distention but has no cardiac symptoms frequently the electrocardiogram alone shows the presence of coronary sclerosis which in the early stages produces only gastro intestinal symptoms in a great many cases. In endocarditis or rheumatic fever the electrocardiogram alone may reveal the participation of the heart muscle. Observations in recent years have disclosed that all of these diseases of the heart even in very advanced stages frequently produce no change either in the size or sounds of the heart so that the electrocardiogram alone makes the correct diagnosis possible. In this connection it is important to realize that in certain acute diseases of the myocardium the appearance of the T waves may change very rapidly and within a short period. This behaviour is found in acute infectious diseases in pneumonia in the rheumatic diseases of the myocardium in coronary sclerosis and thrombosis etc. A single electrocardiographic tracing does not suffice: *in order to reach a positive decision often daily electrocardiograms must be recorded for a long time.*

If during lobar pneumonia or in the course of many bronchopneumonias or after an apparently harmless bronchitis tonsillitis infectious disease etc. the condition of the heart is watched one may be surprised by the extent of the transient alterations which are sometimes found in the electrocardiogram. In cases of rheumatic fever electrocardiographic examinations when repeated daily for a period have also shown that the myocardium is affected at least temporarily in the course of every rheumatic disease. These alterations are ascribed to foci of myocardial inflammation and possibly to vascular lesions which in the course of the rheumatic myocardial diseases lead to the occlusion of the small vessels. A similar situation prevails in periarteritis nodosa.

The majority of abnormal electrocardiograms taken during and shortly after tonsillitis actually may be a result of myocarditis or they may be caused by toxic influences.

In acute *nephritis* the electrocardiogram is often changed. The alterations are usually found during the acute stage and subsequently recede. They rapidly appear and disappear. Ordinarily the initial complex is unaltered while the T wave in Lead I is frequently inverted or invisible, in Lead III the T wave may

become more positive than before the onset of the disease Chest leads may show striking changes

It is very probable that these alterations are induced by vascular spasm which accompanies acute nephritis and by the acute strain to which the left ventricle is first subjected Allergic and toxic phenomena also play a rôle

The appearance of inverted T waves soon after smoking, a regular occurrence in some patients, has been observed by some investigators (Starr, Graybiel and White), and has great practical significance However, it is by no means a common occurrence

It has been known since Einthoven that abnormal T waves may indicate myocardial disease But it was soon discovered that normal T waves may in some instances persist despite severe myocardial disease , moreover, since the great sensitivity of the T waves to external influences has been realized, an unduly critical attitude going far beyond what is necessary has developed If due consideration is given to all the facts cited above, if it is remembered that the appearance of alterations of the terminal deflection or otherwise depends upon the location and extent of the foci in the myocardium, if it is not forgotten that only foci located at certain places may flatten or invert T waves while those situated in other areas may produce perfectly formed positive T waves which may be indistinguishable from normal T waves, if attention is paid to all the extrinsic factors which may alter the T-wave (hypertrophy, digitalis, status of the skin) and if one has learned to evaluate the findings only in conjunction with the clinical symptoms, then the electrocardiogram furnishes incomparable evidence for the appraisal of the myocardium

A prognosis should never be based on the alterations of the terminal deflection alone without consideration of the clinical data Moreover, the most profound inversion of the terminal deflection may disappear entirely and the patient may live his entire life without cardiac complaints or signs of cardiac disease Here also the electrocardiogram merely supplements the results of clinical examination

Alterations of the Terminal Deflection from Digitalis

Alterations of the terminal deflection after the administration of digitalis were first observed by Nicolai and Simons and were studied by A E Cohn Since that time an enormous amount of literature has been written on the clinical and experimental aspects of this subject As the result of premature conclusions, unjustified generalizations, and through animal experiments performed under

result of digitalis, still more displaced in the same direction (Winternitz)

Sometimes these alterations are limited to the first lead, sometimes to the third, but very often *they are present in the same* direction in all leads They may appear in normal individuals as well as in cardiac patients and in decompensated cases may occur even after very small doses of digitalis, whereas they may fail to appear after large doses The presence or absence of alterations in *the electrocardiogram does not give any indication as to the amount* of digitalis previously administered At times the terminal deflection changes its form as early as twenty four hours after the administration of three doses of 0 1 gm (1½ grs) of assayed digitalis leaves, on the other hand, sometimes changes fail to appear even after

a b

FIG 55a and b In a auricular fibrillation with low positive T waves is present, in b after treatment with digitalis sinus rhythm appears Note inverted T waves and depressed S–T segments The P waves are very large

0 5 gm (7½ grs) have been given daily for two weeks The alterations may persist as long as three weeks after the discontinuing digitalis

The tracing reproduced in Fig 55a was obtained from a patient with mitral stenosis There was auricular fibrillation and the *action of the ventricles was fast and arrhythmic But the form of the* initial and terminal deflection was normal

After six days of treatment (three suppositories of digitalis daily, each containing 1 5 cat units), sinus rhythm appeared (Fig 55b) The P-waves are broad and large As the result of digitalis the conduction time is prolonged to the upper *limit of normal (0 20* second), in Leads I and II the S–T segments are deeply depressed below the isoelectric line, and the T waves are absent

The factors which are responsible for the appearance or absence of these alterations are still unknown The situation is very com

the myocardial necroses after digitalis are found in the same animals (cats) in which distinct alterations of the electrocardiogram occur as the result of the administration of digitalis In dogs where digitalis cumulates but slightly and produces only minor alterations in the ventricular electrocardiograms the necroses also fail to appear In support of a connection between the myocardial necrosis and the alterations of the terminal deflection is the fact that it is more difficult to obtain in acute experiment the depression of the S–T segment and the inversion of the T wave after digitalis than it is with prolonged administration

It does not mean a great deal if alterations appear in the electro cardiogram after moderate doses of digitalis and the histological examination fails to disclose any necrosis The alterations must be *severe* before they are histologically demonstrable There are many transitional disturbances and a wide variety of myocardial injuries are not susceptible to histological demonstration

With due reservation these observations permit certain con clusions on these relations as they exist in man and seem to give support to those investigators who advise to conduct digitalis therapy in such a manner that as few changes as possible appear in the terminal deflection

After the administration of large doses of digitalis which had evoked changes in the terminal deflection we have repeatedly observed a distinct acceleration of the red blood cell sedimentation It should also be emphasized that similar alterations of the terminal deflection only appear after relatively larger doses of the more transiently acting strophanthin *If one wishes to obtain definite therapeutic effects without producing changes in the terminal deflections of the electrocardiogram success is more easily achieved with stroph anthin than with digitalis* It is possible that these findings and considerations will lead to a situation wherein strophanthin will be preferred whenever larger doses of digitalis are necessary

The glucosides from *Bulbus scillæ Helleborus niger* etc cause similar changes in the electrocardiogram

Changes in the Electrocardiogram produced by the Upright Position (Standing and Sitting)

In addition to the changes in the electrocardiogram caused by a change of posture which were discussed on p 40 another modifica tion of the tracing dependent upon a change of position is known and possesses great practical importance

The electrocardiogram of some asthenic patients while perfectly

even in the sitting position (Akesson), the electrocardiogram *must always be recorded in the recumbent position* In the early years of

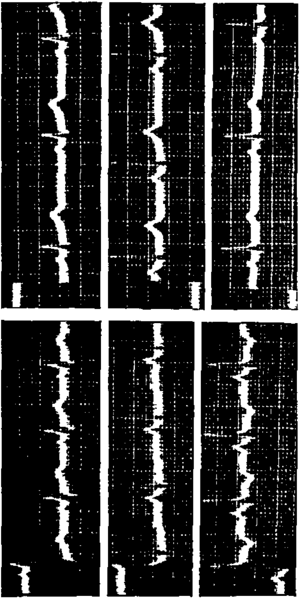

Fig 77 The electrocardiogram taken in a sitting patient (a) and in the same patient lying flat (b)

a b

electrocardiography records were usually obtained in the sitting position.

The importance of this rule, so frequently neglected even at present, is proven by the following observation

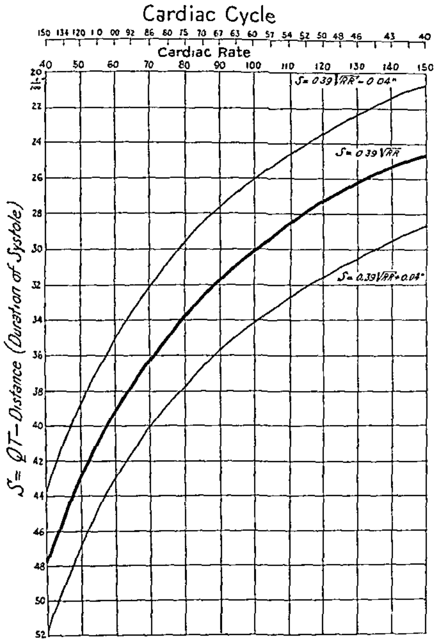

FIG 58 Diagram showing the limits of the normal Q T interval (after Hegglin and Holzmann)

This is expressed most clearly by the shortening of the length of the S–T interval (see p 23) This interdependence is used whenever *the duration of systole is expressed in relation to the rate* The formula which has become most widely known for the calculation

the myocardium The electrocardiographic changes however are not frequent and for this reason can scarcely be evaluated They represent only an incidental finding

With calcium deficiency one sees mainly a prolongation of the horizontal S–T segment and a normal but often a very small T wave In the second group of cases with myocardial diseases the prolongation of systole occurs chiefly through the widening of the T wave itself Hegglin and Holzmann attribute the alteration first mentioned to a prolongation of the duration of excitation and the widening of the T wave in the second form of prolonged systole is ascribed to retardation of the recovery process

Fig 59 shows a patient with a very unusual prolongation of systole It was obtained from an eighty four year old woman with hemiplegia and signs of a generalized atherosclerosis The blood calcium was 8 5 mg per cent

The electrocardiogram shows regular sinus rhythm with a normal initial complex (levocardiogram) In Leads I and II a very wide and deeply negative T wave follows a long normal S T segment In Lead III the T wave is diphasic In the chest lead (left arm—cardiac apex) the initial deflection is normal the T wave is inverted in an unusual manner The Q T distance amounts to 0 60 second the length of one cardiac period is 72 the normal value (0 39 $\sqrt{72} \pm$ 0 04) amounts to 33 09 $\pm$ four hundredths of a second the value of 0 60 lies beyond the limits of the normal of Fig 58

If there is hypocalcemia the intravenous injection of calcium shortens the Q T distance

A shortening of the duration of systole occurs after digitalis and in hyperparathyroidism (see p 112) In the latter instance it vanishes after operative removal of the parathyroid tumour

THE ELECTROCARDIOGRAM IN ABNORMAL FUNCTION OF THE ENDOCRINE GLANDS AND IN THE AVITAMINOSES

In recent years electrocardiographic alterations have been described in association with dysfunction of the endocrine glands In some instances these changes are very characteristic and may have diagnostic significance Our knowledge of these problems is still very rudimentary and one may expect further enlightenment in the near future

Thyroid

The electrocardiogram in *myxœdema* was described on p 44 Similar tracings can be obtained in cretinism

tions have been made in cases of spasmophilia of children (Aschenbrenner and Bamberger)

In contrast to this, a very short Q–T distance and a T wave beginning immediately after the end of the initial deflection has been found in hyperparathyroidism (Ballin, Kellogg and Kerr) The electrocardiogram becomes normal after operative removal of the parathyroid tumour

Pancreas

There is no electrocardiogram characteristic of hypofunction of the islets of Langerhans It is well known that patients with diabetes very frequently have coronary sclerosis and consequently show corresponding changes in the electrocardiogram As a matter of fact the electrocardiogram of diabetic individuals exhibits alterations very early, even at a time when the symptoms and signs of cardiac disease are absent *It is advisable to examine diabetics by means of the electrocardiograph once or twice a year in order to institute the necessary restrictions at an early date after the appearance of the first alterations* Hypo insulinism itself does not produce effects, normal electrocardiograms may be obtained despite the highest known values of blood sugar

The situation is different with hyperfunction of the islets and *the resultant hypoglycæmia* *In this instance profound changes* may be seen in the electrocardiogram

In hypoglycæmia one finds depression of the S–T segments and very low or inverted T waves The initial complexes may become broader and arrhythmias appear These alterations in the electro cardiogram are not always present and at times may fail to appear although the blood sugar values may become astonishingly low (below 30 *mgms per cent*) On the other hand changes in the electrocardiogram may be found at a time when the blood sugar has still not reached its nadir, and may disappear even before the level rises It seems that it is not the insulin *per se* which produces the changes, but the hyperadrenalinæmia consequent upon the injection of insulin *All hearts do not react in an identical manner*

Since the recent introduction of insulin shock therapy in schizo phrenia these investigations have been made in a vast number of individuals who did not have cardiac disease

The cardiac patient may react much more strongly to the injection of insulin than the healthy individual Repeatedly we have been able to convince ourselves of this fact since even very small doses of *insulin (15–20 units) which failed to produce a noteworthy fall* of the markedly elevated blood sugar, were able to precipitate severe

anginal attacks and produce marked alterations in the electrocardiogram. For this reason the employment of insulin requires great caution in patients with coronary disease. Cases in which injury to the myocardium has been observed following moderate doses of insulin are rare but equivocal observations have been made suggesting the employment of insulin only in those cases of coronary sclerosis in which the metabolic situation unconditionally demands it. There are cases in which one would prefer a continuously high blood sugar to an insulin injury.

Marked changes are also observed in the electrocardiogram during the metrazol (cardiazol) shock treatment of schizophrenia.

Ovaries

In some cases of ovarian insufficiency with hyperexcitability of the heart, tachycardia with or without hypertension, anxiety, palpitation, precordial pain and manifestations of hyperthyroidism, distinct changes in the terminal deflection of the electrocardiogram are frequently found. These changes consist primarily in a depression of the S-T segments in all leads and therefore, according to the rule of Einthoven, they are most pronounced in Lead II. The T waves may also be low or absent; rarely are they inverted.

Fig. 60. Depression of the S-T segments in hypertensive tachycardia in the climacteric.

The electrocardiogram in Fig. 60 was recorded in a patient with a climacteric hypertensive tachycardia. The blood pressure varied between 180/60 and 220/75, the heart rate between 100 and 140. Distinct signs of hyperthyroidism were evident and the basal metabolism was increased to plus 70. The heart was not dilated. The electrocardiogram, Fig. 60 (the patient had not received any treatment at the time), shows a deep depression of the S-T segments in all leads, most marked in Lead II. All other waves, including the T waves, are normal. At the present time the electrocardiogram is normal.

This electrocardiogram is characteristic and can easily be distinguished from the type of record usually obtained in cases of hypertension which shows a levocardiogram and oppositely directed terminal deflections in Leads I and II. Likewise the electro-

cardiogram in hyperthyroidism has a different appearance (see p 111)

The same alterations also are observed in the absence of hypertension and without signs of hyperthyroidism

Fig 61 reproduces the electrocardiogram of a forty six year old woman who consulted a physician because of substernal pain of several weeks duration and swelling of the feet For one week she suffered from a dull pain in the region of the lower sternum Three years earlier an artificial climacterium was produced by bilateral oöphorectomy A very mild hyperexcitability existed physical examination *revealed a normal circulation* blood pressure amounted to 135/65

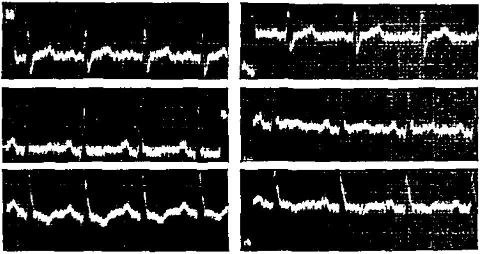

Fig 61 The electrocardiogram of a climacteric patient before (Fig 61a) and after (Fig 61b) treatment with estrogenic hormone

The electrocardiogram in Fig 61a shows, *in addition to a sinus* tachycardia depressed S T segments and absence of the T waves in Lead II Nine days later (Fig 61b) the rate already had become slower and the T wave of Lead II positive During this interval the patient daily had received 2 000 units of estrogenic hormone in the form of injections No other therapy was employed except sedatives Her symptoms disappeared completely

The same alterations may be encountered in young women with *signs of ovarian insufficiency (hypoplasia of the genitalia)* It must also be emphasized that these alterations may appear many years before as well as many years after the menopause, the menopause and the manifestations of the climacterium often do not coincide Women who suffer from "hot flashes" do not always show these

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THE ELECTROCARDIOGRAM IN CORONARY THROMBOSIS

The expansion of our knowledge concerning the clinical picture of coronary thrombosis constitutes one of the great advances which has occurred in the realm of cardiac diseases in recent years In the main this has been made possible by electrocardiography The electrocardiogram permitted the diagnosis in the common atypical varieties of coronary thrombosis which formerly were erroneously regarded as pleuritis cholelithiasis gastritis subdeltoid bursitis as well as in cases without pain or without typical pain When these cases of coronary thrombosis became with considerable certainty recognizable in their early stages the symptomatology of the clinical picture underwent great development so that as a rule the diagnosis can to day be made without an electrocardiogram simply on the basis of the clinical symptoms But even at present cases are periodically encountered in which the electrocardiogram alone provides the evidence This is particularly true when some time has elapsed since the thrombosis occurred or when all the clinical signs of cardiac disease are absent and the history is atypical

The first experimental work on the electrocardiogram of coronary thrombosis was initiated by Smith the first clinical electrocardiographic investigations were performed by Herrick and Pardee (1920)

If in a case of coronary thrombosis an opportunity occurs of recording the electrocardiogram soon after the beginning of the attack and of taking repeated records *during the subsequent course very characteristic alterations may be observed in a certain percentage of the cases*

As the first sign one observes the terminal deflection merge into the initial deflection (Fig 62) While a long or short S–T segment is normally observed between the two sections of the ventricular electrocardiogram, this is absent during or immediately after the attack of pain or in the absence of pain after the beginning of the thrombosis The descending limb of the R wave does not return to the isoelectric line, but merges into a highly elevated final deflection, which after a horizontal course of variable length returns

to the zero line in an outward convex arc. In rare instances this interval takes its origin from the apex of the R-wave so that such ventricular complexes may be called purely "monophasic electrocardiograms." The T-wave often merges in the high arched S-T segment and cannot be demarcated. This is called a "high take-off" of the terminal deflection from the initial complex. This change may be most marked in Lead I or Lead III If it occurs in Lead I, the opposite changes *may* occur in Lead III (Parkinson and Bedford), that is, there may be a low take-off of the S-T segment

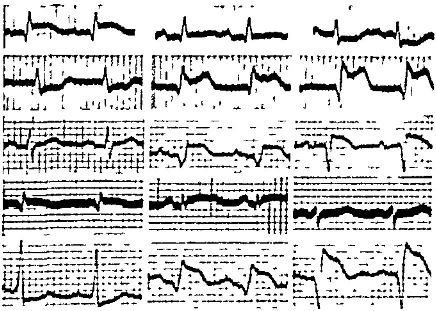

FIG. 62 The electrocardiograms of five cases of coronary thrombosis shortly after the beginning of the symptoms.

from the ascending limb of the S-wave in Lead III. If there is a high take-off in Lead III, a low take-off may be noted in Lead I and *vice versâ*, however, the latter is often absent.

Fig 62 shows the electrocardiogram of five cases of acute coronary thrombosis, all records were obtained within a few hours after the beginning of anginal pain

In the first and fourth series, a high take-off is present in Lead I. The alterations are most distinct in the first series because the initial deflection is larger, but the upward shift of the S-T segment is also visible in the fourth series and it passes into an inverted T-wave In the first series Lead III exhibits a mirror image of

Lead I ("low take off") In the fourth series Lead III of the electrocardiogram is normal

In the second third and fifth series the high take off is present in Lead III while in Lead I of the second and fifth series a low take off may be observed Lead II also shows more or less marked changes in all cases

The deep Q waves in Leads III and II are noteworthy in cases 2 3 and 5 In most of these cases the P waves are abnormal (broad split and low) a finding to which Master has directed attention The absence of widening of the initial deflection is common

At times these electrocardiograms persist for only a few minutes more commonly they last for hours less often for days If one follows the retrograde change it will be noted that the terminal

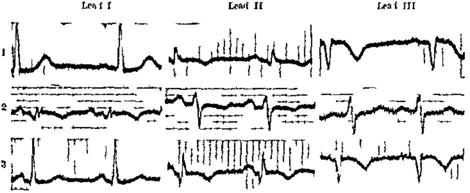

FIG 63 Three electrocardiograms of three patients who had suffered from coronary thrombosis several weeks earlier

deflection takes off from the descending limb of the R wave at successively lower points and an increasingly negative T wave follows until the S–T segment finally lies approximately on the isoelectric line in the form of an upwardly convex arc followed by a deep inverted ("cove shaped") T wave These electrocardiograms which were designated by Pardee as "coronary T waves" are found either in Lead I or in Lead III and at times a high positive T wave may be seen in Leads III or I Such electrocardiograms may persist for weeks or months

In Fig 63 a series of curves is reproduced from patients in which the coronary thrombosis had occurred four to six weeks previously In the first and third series the changes are most distinct in Lead III in the second series in Lead I The deep pointed T waves are evident in all cases and are preceded by short S–T segments which are slightly curved with the convexity directed upwards

Gradually the inverted T wave becomes smaller and after some weeks disappears entirely until finally a normal positive T wave reappears and becomes more and more distinct. In certain cases the return of the normal T wave may require years. If the changes have progressed this far the tracing cannot be distinguished from a normal electrocardiogram. In some cases observed for many years the electrocardiogram remains permanently abnormal despite a complete clinical recovery.

Fig. 64 shows the electrocardiographic alterations in a fifty-six-year-old man who had had a thrombosis of the *Ramus descendens*

Lead I | Lead II | Lead III

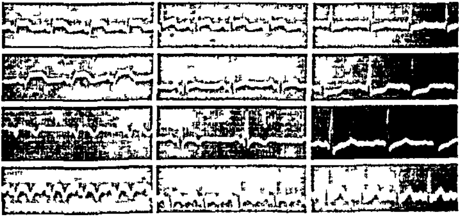

Fig. 64. A series of tracings from a case of coronary thrombosis (the three leads have been placed alongside of each other)

deflection) Fig 65b shows the electrocardiogram after the lapse of three months The T wave is still slightly negative in Lead I A short time later the electrocardiogram became normal and to day, four years later, *the patient follows his occupation as a physician, free from symptoms and with normal cardiac findings*

All the illustrations reveal that the alterations of the terminal deflection (high take off, etc) are in some cases most marked in Lead I, in others in Lead III By means of exact anatomical examination of clinically well observed cases Barnes and Whitten were able to demonstrate that the various leads present different *alterations in accordance with variations in the site of the thrombus*

a b

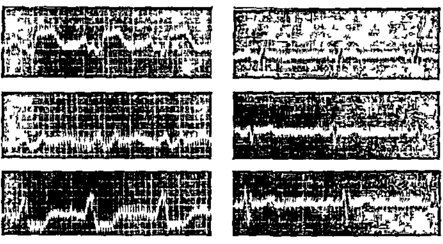

Fig 65a and b In (a) the electrocardiogram of a recent coronary thrombosis in (b) the tracing of the same patient three months later Fig 65 is deformed by an alternating current

If the high take off or the ' coronary T ' is most marked in Lead I (T_1 type) there is a thrombosis in the *ramus descendens* of the left coronary artery and infarction of the anterior wall of the left ventricle exists If the alterations (high take off, inverted T) are found in Lead III (T_3 type) this indicates an occlusion of the ramus descendens of the right coronary artery (posterior wall *infarction) the thrombosed branch of the ramus descendens* posterior supplies the posterior wall of the *left* ventricle Infarction of the right ventricle is rare

In the course of time exceptions to this rule of localization have been noted, they can be explained partly by anomalies in the course of vessels and by the position of the heart but *chiefly by the participation of other coronary vessels* In a majority of cases

A number of other alterations of the QRS complex in coronary thrombosis have been described by various authors but those changes are not characteristic and are very inconstant All the factors which produce exceptions to the rule of the form of T_1 or T_3 in anterior and posterior infarction also create exceptions in the form of the initial complex indeed *frequently the initial deflections tend to be modified since in addition to the thrombosed artery other branches of the coronary vessels are also commonly affected*

The following point deserves special emphasis the electro cardiogram just described alone and without other evidence from the history and without other signs does not justify the diagnosis of 'coronary thrombosis The electrocardiographic alterations which have been discussed are the result of a myocardial injury precipitated by the thrombosis and are not produced by the thrombus itself For this reason it is comprehensible that myocardial injury from other causes which leads to the elimination of a large area of muscle will occasionally produce the same changes Thus the same kind of electrocardiograms that occur in coronary thrombosis may at times be encountered in a post diphtheritic myocardial lesion in myocardial injuries during the course of pneumonia (p 133) in which oxygen deficiency and myocarditis may co exist and in severe myocarditis of other types In the next two sections it will be pointed out that very similar pictures may be seen in pericarditis and pulmonary embolism High take off of a degree commonly *encountered in coronary thrombosis is occasionally noted for a* few minutes after exercise in patients with advanced coronary *stenosis*

Since this situation prevails it must be concluded that the electrocardiograms described *in coronary occlusion (usually throm* bosis rarely embolism and only very rarely other causes such as occlusion by a metastatic tumour which compresses the artery from without) are not pathognomonic so that the electrocardiogram *alone* should never provide *the diagnosis As in other instances* the tracing may be interpreted only in conjunction with the history and the clinical picture

Only 30 to 40 per cent of the cases of coronary thrombosis present in the electrocardiogram the typical alterations described The absence of these changes does not militate against the diagnosis In many cases the alterations are not characteristic The T waves may be absent or inverted in Lead I or II the S–T segments may be displaced below the isoelectric line the initial deflections may be widened or split *Very rapid indeed daily changes in the electro*

cardiogram are not unusual Fundamentally any of the electrocardiographic changes which develop in conjunction with other myocardial injuries may occasionally be encountered in coronary thrombosis

At times the electrocardiogram despite repeated tracings remains normal though the patient undoubtedly suffers from coronary thrombosis as subsequently proved at necropsy The term silent area in the myocardium has been introduced to explain this absence of electrocardiographic change Chest leads (p 150) have been added to the three standard leads precisely for these cases At times the electrocardiographic changes appear only several days after the thrombosis of a coronary artery We have observed cases in which the electrocardiographic alterations became apparent only on the tenth or fourteenth day after an attack of precordial pain which was accompanied by a transient rise of temperature A single electrocardiographic tracing does not suffice

An explanation for the appearance of the ' high take-off ' of the S–T segment still presents difficulties Several investigators have attributed these curves to the so called injury current thus they are compared to electrocardiograms which are obtained in animal experiments when the tissues below *one* electrode are severely injured a direct lead from the heart being employed In this instance the tracings assume the same monophasic character which in many cases is noted soon after the onset of coronary occlusion Others explain the upward displacement of the S–T segment by the necrosis of a large area of muscle and the resultant disappearance of its potentials A decided deficiency of oxygen and the consequent metabolic disturbances or the premature occurrence of deactivation are also considered to be causes of these remarkable curves The first explanation seems to have most support

THE ELECTROCARDIOGRAM IN ACUTE PERICARDITIS

The electrocardiogram which develops in conjunction with a pericardial effusion without inflammatory phenomena has been described on p 43 and the tracing associated with pericardial adhesions on p 39

Very frequently one finds remarkable electrocardiographic changes in exudative pericarditis (Scott Feil and Katz) These alterations are important because they may simulate the changes caused by coronary thrombosis

Here also several stages are distinguished (Herrmann and

Schwab) In the first acute stage one finds a high take off similar to that of coronary thrombosis As a matter of fact it rarely attains the high *degree encountered in cases of* coronary thrombosis but it may be very distinct However in acute pericarditis the alterations of the S–T segment are perceptible in *all* leads whereas in coronary thrombosis these changes are demonstrable only in Lead I (and II) *or* Lead III (and II) Very often it does not actually involve a high take off of the terminal deflection but merely an upward displacement of the S–T segment The R wave returns to the isoelectric line but immediately afterwards the S–T segment rises abruptly The T wave remains positive so that the S–T segment is frequently curved with its concavity directed upward

Fig 66 shows a series of tracings from a twenty four year-old woman whose pericarditis was clinically evident In Fig 66a the elevation of the S–T segments is distinct Since it is present in all leads it is according to Einthoven s rule, most distinct in Lead II The Q wave of coronary thrombosis is absent Two days later the changes were much more obvious (Fig 66b) The initial deflection is widened the descending limb

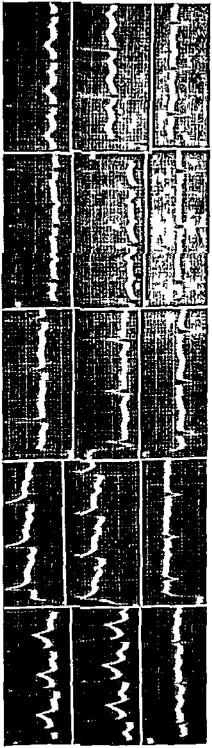

Fig 66a—e A series of electrocardiograms of a case of rheumatic pericarditis

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by the pressure of the exudate on the superficial layers of the myocardium with consequent anæmia of the muscle must be supplemented by the observation that the myocarditis which accompanies the pericarditis produces the abnormal electrocardiograms (Vander Veer and Norris).

The alterations which develop in various forms of pericarditis (rheumatic, tuberculous, uræmic) resemble each other. In most if not all, instances there is an accompanying pericarditis when a high take off is noted in the course of pneumonia

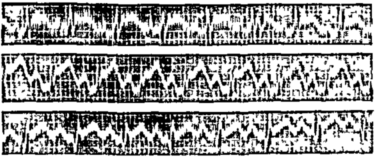

FIG. 68. The electrocardiogram of a case of pericarditis which occurred in conjunction with coronary thrombosis

Recent unpublished experiments of the authors show that chemical or mechanical irritation of very small areas of the epicardium and of the myocardium beneath, cause a high take off and a monophasic electrocardiogram. This is explained by a current of injury. It is noteworthy that very profound changes of the electrocardiogram can be induced by irritation of extremely small areas and it is equally interesting that *the electrocardiographic alterations are the same regardless of the site of epicardial irritation.*

THE ELECTROCARDIOGRAM IN CARDIAC INJURY AND TRAUMA

Distinct electrocardiographic alterations appear in trauma with and without direct injury to the heart

Fig. 69 was obtained from a patient who in an attempt at suicide shot himself in the right ventricle *On fluoroscopic examination the bullet was found freely movable in the right ventricle. At operation the wound of entry in the anterior wall of the right ventricle was closed.* None of the larger branches of the coronaries was ligated.

An embolus in a small branch of the pulmonary artery may constitute a dangerous complication even for a patient whose circulation is healthy. But if the patient has cardiac disease such an embolism may precipitate a decompensation which cannot be favourably influenced by cardiac remedies and which may be rapidly fatal.

These dramatic manifestations which are often out of proportion to the size of the relatively small pulmonary area deprived of its blood supply have been studied repeatedly without a satisfactory explanation for their occurrence having been found.

Recently a severe myocardial injury which manifests itself by marked changes in the electrocardiogram has been discovered to

Fig. 70a and b. In (a) the electrocardiogram shortly after the appearance of a pulmonary embolism; in (b) the electrocardiogram—four days later—is negative.

be one of the causes of the rapid cardiac failure (McGinn and White, Scherf and Schönbrunner, Barnes).

A forty one year old woman developed phlebitis after an injection of glucose into a varicose vein of the right leg six months before admission to hospital. Two months after the onset of the phlebitis high fever occurred and a thrombosed varix was noted in the region of the left knee; it was tender to pressure and the skin of the region was reddened. Subsequently a recurrence of acute phlebitis developed in the right leg. The patient sought treatment in hospital because of the marked feeling of oppression in the cardiac region and a periodically recurring præcordial pain of an anginal type. A very slight mitral stenosis without change of either cardiac size or shape was found; merely a short presystolic murmur was audible at the apex. A large thrombotic vein, tender

coronary vessels were *normal* *no myocardial disease was found* which could explain the profound alteration of the electrocardiogram

The electrocardiogram of Fig 71b was transcribed shortly after the beginning of the symptoms of pulmonary embolism two days after Fig 71a The distinctly low take off of the terminal deflections from the ascending limb of the S waves is noted in Lead I and deep Q waves are evident in Lead III together with a high take off of the S-T segments from the R waves

According to McGinn and White a deep Q wave with a negative T wave in Lead III is very characteristic of the electrocardiogram *of cases of pulmonary embolism* In Lead I an S wave appears and the T wave originates somewhat below the isoelectric line Barnes has given some points for the differential diagnosis from coronary thrombosis which may sometimes present great difficulties

In this connection it must be added that on the basis of many post mortem examinations we believe that deviations from Barnes tabular arrangement are very frequent in coronary thrombosis as well as in pulmonary embolism and that all varieties of T wave changes are encountered in both conditions (Fig 70)

McGinn and White attribute these alterations to a marked dilatation and partial failure of the right heart i e to an acute cor pulmonale It is indisputable that excessive burdens are placed upon the right heart and may play a role But it is equally true that they may also definitely fail to appear In two personally observed cases with distinct electrocardiographic alterations resulting from pulmonary embolism one of us failed to observe any evidence of cardiac dilatation or disease at necropsy From a study of cases of this type *it has been concluded that in pulmonary embolism a* pulmo coronary reflex occurred through excitation of the endings of the pulmonary vagus which induced an inadequate blood supply to the heart especially to the overburdened right heart In this manner the subjective complaints of the affected patient (anginal pain) and the objective clinical findings (gallop rhythm strikingly rapid progressive cardiac failure in the absence of cardiac dilatation) *are explained* Likewise the therapeutic effect of vasodilators (papaverine theophyllin atropine) supports the explanation just advanced Not only experimental findings but also observations like those given in connection with Figs 121 and 148 favour the existence of pulmocardiac reflexes Reflexes to the heart are known to originate not only from the lungs but also from the nose ears eyes throat all abdominal organs and the skin A certain condition of the reflex arc the receptor and the effector organ (the heart) is a prerequisite for the occurrence of these reflexes

off from the ascending limb of the S waves far below the isoelectric line The attack was stopped within twenty minutes by an injection of strophanthin Two hours later an electrocardiogram similar to Fig 72a was recorded

Severe attacks of angina pectoris may appear simultaneously with these alterations in the form of the electrocardiogram In regard to the unpleasant even painful sensations in the precordium in paroxysmal tachycardia it should be mentioned that this kind of distress may appear in patients with otherwise normal hearts It is more common in patients whose susceptibility is increased by co-existing cardiac disease Since in many cases of paroxysmal palpitation the tachycardia itself is not perceived the anginal pain being the most outstanding symptom that may continue for hours the tachycardia may not be detected the real nature of the attack thus escape recognition and the illness may be attributed to coronary thrombosis (Camp and White)

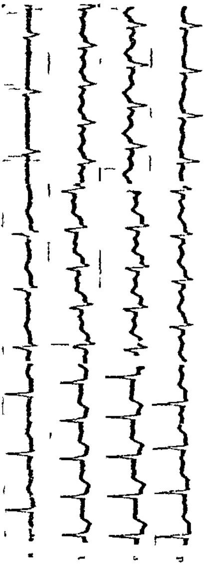

Fig 73—(a) shows the electrocardiogram at rest of a case of luetic coronary stenosis (b–d) a series of tracings (the leads have been placed alongside of each other) showing the marked alterations of the terminal deflection after an injection of atropine

How profoundly a simple acceleration of the heart can alter the electrocardiogram may be seen by a study of cases of marked coronary stenosis of luetic origin

A patient with luetic insufficiency of the aortic valves and attacks of angina pectoris presented an electrocardiogram which was practically normal (Fig 73a upper series) After the intravenous injection of 2 mg ($\frac{1}{33}$ gr) of atropine, the rate of the heart increased

in the ordinary manner they are normal in nearly 60 per cent of the early cases. Moreover when aortitis, hypertension, valvular lesions or a murmur is detected this does not prove that the symptoms for which the patient seeks relief can be properly ascribed to angina pectoris.

The diagnosis is made on the basis of the history alone. If the patient describes the typical complaints, if pain appears after well known occasions, if it radiates in the usual manner, if it is relieved at once by nitroglycerin, one is inclined to assume that *angina pectoris is present. Cases which deviate from the usual* pattern are not uncommon, and at times it is scarcely possible to make a diagnosis with certainty.

Errors commonly occur particularly at the beginning of the disease. In addition it happens that in recent years the history of patients *is no longer unbiased* for every layman is aware of the symptomatology of angina pectoris through instances of the disease in relatives or acquaintances or through descriptions in the newspapers.

Considerable progress has been made in recent years since a way has been found to make possible an early diagnosis in cases of this type, to confirm the diagnosis by objective findings and at *the same time to provide information about the events occurring* in the heart muscle during the attack.

By a number of investigators electrocardiograms have been transcribed *during* attacks of angina pectoris which occurred during rest and were not associated with coronary thrombosis. They *found very distinct alterations of the S–T segments and of the* T waves during the attack (Feil and Siegel). But reports on this event are relatively uncommon since an opportunity of recording an electrocardiogram *in* an attack lasting only for a few minutes is presented in comparatively few cases and then usually by accident.

More recently the recommendation has been made to forestall these rare opportunities. Instead of waiting until it became possible *to record the electrocardiogram during an attack, it was suggested* that in doubtful cases an attack be induced by imposing a burden on the heart by means of physical exertion. The electrocardiogram obtained after exercise could then be compared with the electrocardiogram recorded during rest (Scherf, together with Goldhammer, Hausner, van Muyden). Wolferth and Wood studied the electrocardiogram of patients with angina pectoris after physical *exercise and found marked changes. However they concluded* that this test is without diagnostic value.

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Fig 74 shows fairly well the maximum extent of alterations of the S–T segment which occurs normally after exercise in a patient without coronary stenosis

If the exercise is very strenuous, the alterations of the electrocardiogram may be extreme Electrocardiograms have been repeatedly recorded in marathon runners immediately after the completion of the race and have been found normal But in some *runners after such a race or after skiing for 50 km, in which the* contestants finished in poor condition, negative T waves and invisible T waves have been reported (Ludwig)

It is obvious that such efforts are not comparable with those demanded of patients in the exercise experiment When an indi

Fig 75 Exercise in a case of luetic coronary stenosis, the three leads are recorded alongside of each other

vidual climbs a flight of stairs even very rapidly, not only in the healthy person but also in cardiac diseases of various types (valvular *lesions, myocardial diseases, hypertension), only the normal range* of alterations of the waves, previously described, is noted A T wave which was formerly negative, after exercise may become positive, if previously depressed the S–T segment may become isoelectric

In patients with coronary stenosis the changes of the electrocardiogram are entirely different At times when the electrocardiogram is recorded during rest there are few or no abnormal electrocardiographic changes, *since the heart obtains an adequate amount* of blood despite the coronary stenosis But if a burden is placed

upon the heart by physical exertion or by excitement its oxygen requirement increases however, as the result of the coronary stenosis an increase in the blood supply to smaller or larger areas of muscle is not possible accordingly a disproportion develops between the blood requirement and blood supply and an area of the muscle thus becomes ischemic with the result that the electrocardiogram is changed and pain occurs

Fig 75 An electrocardiogram recorded during rest and at intervals after exercise in a patient with angina pectoris produced by a luetic stenosis of the coronary arteries. The lowermost series of tracings reproduces the electrocardiogram during an attack which appeared during rest in bed (hypertensive crisis)

The patient from whom Fig 75 was obtained was thirty years of age She visited the clinic because for two years she had noted that painful pressure developed behind the sternum after great exertion which compelled her to stop, more recently this developed even when she walked rapidly for a short distance A systolic murmur was audible over the region of the aortic valve and the Wassermann reaction was positive

The electrocardiogram (Fig 75 upper series) registered at rest was normal, only a left axis deviation was present, a loose contact produced an artefact in the tracing in Lead II A marked acceleration of the heart rate resulted from rapidly climbing two flights of stairs The terminal deflections in Lead I (second series) are below the isoelectric line the S-T segments are also lowered in Leads II and III but the T waves are higher than at rest owing to the physiological effect of effort on the electrocardiogram After five minutes

(third series) the S–T segments were still depressed below the zero line in all leads and the T waves were very low Ten minutes after the exercise the electrocardiogram was still not entirely normal (lowest series)

The electrocardiogram in Fig 76 shows a typical positive exercise test in a patient who suffered from an insufficiency of the aortic valves At rest the electrocardiogram was normal except for a very slight depression of the S–T segments in Leads I and II (first series) One minute after climbing a flight of stairs 18 metres in height a deep depression of the S–T segments appeared in Leads I and II and the T waves in Leads II and III became negative (second series) Two minutes after exercise the electrocardiogram was still unchanged (third series) after five minutes a slight return towards normal is evident (fourth series) The electrocardiogram did not become normal until one half hour later (fifth series)

The T waves were then even higher than before a finding which is not rare and still not entirely explained T waves which before effort were invisible or inverted may become normal for some time after the pathological reaction to effort has subsided

The lowermost series in Fig 76 shows the electrocardiogram during a spontaneous attack of angina pectoris which appeared during rest it was accompanied by a decided increase of blood pressure (hypertensive crisis) The tracing shows the same alterations as those appearing after physical exertion This finding is also noted regularly (more than forty personally observed cases) *The electrocardiographic alterations in angina pectoris after exercise always resemble those which are recorded during a spontaneous attack*

The series of tracings in Fig 77 were obtained from a patient who a few months later showed at necropsy a marked syphilitic stenosis of the coronary artery orifices The symptoms which caused the patient to attend the clinic were obscure and the diagnosis was uncertain The electrocardiogram recorded after rest showed *nothing abnormal Only a levocardiogram was present After slowly* climbing a flight of stairs 18 meters in height extreme alterations appeared in the terminal deflections together with deep T waves (second series) After five and ten minutes (third and fourth series) *there was a slow return to normal but twenty minutes elapsed* before the electrocardiogram returned to its original status

The alterations which are found transiently after exercise in patients with coronary stenosis usually affect the S T segment and the T wave particularly, *more exceptionally* the initial deflection is affected as well The S–T segment is usually depressed below

the isoelectric line but only those tracings are considered positive in which the depression is *distinctly* more marked than in normal individuals. At times this depression is present in all leads but in

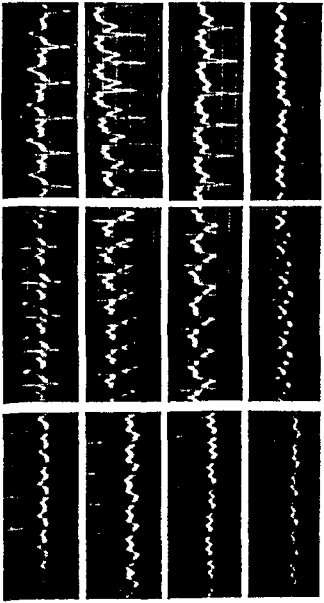


FIG. 22. (The three leads alongside of one another.) The uppermost series shows the electrocardiogram at rest, the remainder after climbing stairs; the patient had a large coronary sclerosis.

other cases is noted only in Lead I or only in Lead III. In rare instances an elevation of the S–T segment appears in one of the leads. Likewise a transient inversion of the T wave may be found

in one or several leads The alterations of the initial deflection after exercise are less characteristic and, at times, may be produced by a change in the position of the heart, consequent upon changes in the position of the diaphragm Occasionally a high take off like that of coronary thrombosis may appear for a few minutes

The electrocardiogram must be recorded, not only immediately after exercise, but also one, two, five, and ten minutes afterwards At times the changes are manifest *only* immediately after exercise, that is, they are extremely transient In other cases only the normal increase in the height of the T waves is noted in the electro cardiogram transcribed immediately after the effort the depression of the S–T segment or the inverted T wave may appear only after some minutes have elapsed The chief offence committed against this method of investigation lies in the failure to record the electro cardiogram five and ten minutes after effort and relying upon a single record registered immediately upon completion of the exercise

In many cases the electrocardiographic alterations persist for a long time—for example, as long as thirty minutes In some instances they are still distinct although the pain has long since vanished In other words, they are independent of the pain *Not rarely distinct alterations in the form of the electrocardiogram are observed although the patient experienced no pain during or after the effort* Likewise when the pain quickly disappears (after nitrites) the electrocardiographic alterations may persist for a considerable time

A negative exercise test, that is, the absence of the changes after effort is against a marked disturbance of the blood supply to the heart but not against stenosis of a coronary ostium Moreover, occasionally a normal exercise test is observed in aortitis despite complete occlusion of *one* coronary ostium In this instance the stenosis is " compensated " by extensive anasto mosis with the other coronary artery It is also possible that a stenosis in the region of some smaller branches of a coronary artery will not produce distinct alterations in the form of the electrocardio gram after exercise However, a positive result of the test can be employed with certainty Abnormal reactions to exercise are most common and most evident in aortitis which has led to a stenosis of the coronary orifices But they are not rare in coronary sclerosis

It is understood that the performance of the exercise test should not be dangerous for the patient Accordingly, the exercise must be adapted to the condition of the individual subject Since

patients who consult physicians for obscure complaints must in large cities climb stairs during every working day, since they report that their symptoms appear only after *physical exertion*. It may be assumed that the amount of exercise arranged by the physician will bring no greater danger than that performed several times each day by the patient. *For this reason no greater effort should be demanded from the patient than that which he undertakes daily.* A patient who feels the pain at rare intervals and only after great exertion and as the result of several factors (after meals walking in cold weather) may accordingly be asked to climb two flights of stairs and under certain conditions even four or more. If the patient complains of pain after slight exertion, it may suffice for him to perform knee bending exercises a few times or to rise and lie down several times in order to produce abnormal changes in the T waves.

Since there is absolutely no parallelism between the appearance of pain and the electrocardiographic changes the alterations described are a measure of coronary perfusion alone and not of angina pectoris. All clinical and experimental findings obtained up to the present time show that the temporary changes described and illustrated in the electrocardiograms after exercise appear only in disturbances of blood supply to the heart. Among other things this is proven by the observation that the pathological changes after exercise are as a rule absent when the patient performs the work immediately after the administration of nitroglycerin.

The alterations after exercise described in the terminal deflection are not found in cardiac patients of the most diverse types unless coronary stenosis is present (Wood and Wolferth, Scherf and Goldhammer). In rare instances they are observed in myocarditis.

The experimental observations of Dietrich are important in regard to the appraisal of the disturbances of myocardial blood supply after exercise in cases of coronary stenosis; he found experimentally that when the ratio between coronary blood supply and cardiac performance was only three fourths of normal a pointed *inverted* T wave and an abnormal S-T segment appeared.

When a patient breathes from a closed spirometer (Rothschild and Kissin) or in a low pressure chamber or breathes mixtures of nitrogen and oxygen that are poor in oxygen (Dietrich and Schwiegk) as soon as marked anoxemia (and consequent anoxia of the heart) is produced exactly the same alterations of the electrocardiogram are seen. Since these alterations are evoked in healthy individuals if oxygen deficiency is continued sufficiently long and since a special apparatus is necessary, this method does not lend itself

readily to routine practice Clinical and experimental observations support the suggestion that it is not the oxygen deficiency alone but the metabolic disturbances in the cardiac muscle created by it which evoke the changes in the electrocardiogram

The advantage of a correctly performed exercise test for the clinical study of angina pectoris consists primarily in the fact that through it an objective diagnosis has become possible and the physician is not compelled to rely only upon the subjective symptoms of the patient With the electrocardiogram it is often possible to make the diagnosis in atypical cases at the beginning of the disease when pain (pressure and oppression) appears at infrequent intervals and is neither precipitated by the usual factors nor radiates in the typical manner Every physician can recall patients in whom for a time at least he doubted whether or not the ominous *diagnosis should be made* It should be emphasized once again that only the positive exercise test the actual appearance of the changes in the electrocardiogram is decisive

Unfortunately the physiological depression of the S–T segment which is often present as in Fig 74 is regarded as pathological by some physicians Considerable caution should be exercised in the interpretation of tracings and only responses to exercise which are unequivocably abnormal should be designated as pathological and not borderline cases

The registration of an electrocardiogram after exercise also permits the physician to evaluate therapeutic measures, patients with coronary stenosis may show the most extreme electrocardiographic changes after exercise but if the effort is undertaken soon after the injection of theophyllin or papaverine very often slight or no changes develop in the tracing By means of electrocardiograms after exercise the progress or disappearance of a disturbance of blood supply in the cardiac muscle can be appraised with greater certainty than by any other method

No *prognostic* conclusions should be drawn from the outcome of the exercise test A patient may show a normal electrocardiogram after exercise to day and to morrow may suffer from coronary thrombosis in other cases despite distinctly positive exercise tests a progressive amelioration of symptoms and the gradual development of a normal exercise response must be ascribed to the gradual dilatation of collateral vessels But if the patient consults a physician for trifling or atypical complaints and the profound changes of Figs 75–77 are discovered the patient should be warned about the situation The practice of recording electrocardiograms

and inconvenient for the physician Patients suspected of a recent coronary thrombosis should not be allowed to assume a sitting position Wilson (1930) had called attention to the fact that if one electrode were placed near the heart, the second electrode could be applied at any optional area without essentially altering the electrocardiogram, *therefore it may be applied to an arm or leg*

For this reason connexions were made from the region of the apex beat or from the fourth left intercostal space near the sternum (region of absolute cardiac dullness) and from the left leg or the right arm, the latter is better (Roth) because it yields larger P waves Since there is already one electrode for the right arm *in the standard leads, only one additional electrode is required for the region of absolute cardiac dullness*

Under these conditions normally one obtains a negative P wave, a distinct Q wave followed by an R a slightly depressed S–T segment and a negative T wave With this type of lead however, the most important wave the T wave normally is inverted This situation presents considerable difficulty to the beginner, because—in contrast to the standard leads—the positive T wave is abnormal while the negative T wave has to be considered normal, moreover, the presence of a Q wave is physiological It has therefore often been suggested to reverse the polarity in order to make the tracing resemble the customary picture Recently *a committee of the American and British Heart Associations made the recommendation* to lead from the left leg or the right arm to the heart so that—as in leads from the extremities—positivity under the electrode applied to the cardiac region yields upright excursions and a negativity downward deflections Therefore to record the chest lead the electrode ordinarily destined for the left arm is applied to the cardiac apex or just to the left of the lower end of the sternum, and the electrode for the right arm remains at its usual place, the apparatus is switched into position as for Lead I

Under these conditions the normal ventricular complex is similar to the normal ventricular complex of the standard leads One observes always as in Fig 78 (upper series first tracing from the right) a positive (rarely isoelectric or inverted) P wave, an R-wave which is followed by an S, a slightly elevated S–T segment, and a positive T wave

The absence of an R wave or an R under 2 mm in height, widening of the initial deflection, depression of the S–T segment below the isoelectric line or marked elevation above it, or the absence *of a positive T wave is considered pathological*

In *young* individuals (up to the twentieth year of life and in exceptional cases beyond) we have also observed inverted T waves in the *chest leads although cardiac examinations were continually normal*, and there was no evidence of cardiac disease For this reason the discovery of an abnormal T wave must even in older patients be *evaluated with caution for it may be encountered, though rarely, in* healthy older individuals The absence of an R wave was interpreted as a much more significant and reliable sign for the diagnosis of a myocardial disease In general, even an R wave which has an amplitude of less than 2 mm is regarded as pathological But it is certain that R waves of this size also occur normally In order to interpret a chest lead it is extremely important to know the site from which the lead was taken, even a small shift of the electrode near the heart may produce very marked changes in the electrocardiogram

At present the leads are taken from the two areas mentioned First, from the region of absolute cardiac dullness left parasternally, and second from the cardiac apex If one gradually moves the chest electrode from the left lower parasternal region towards the apex, then the alterations which appear are fairly regular If by chance, the left parasternal electrode is applied somewhat higher, the R wave may vanish The amplitude of the R wave is much reduced in a lead from the anterior chest wall in the left parasternal region in comparison to one taken from the cardiac apex Just as the R wave increases in size as the electrode is moved from the left lower sternal border toward the cardiac apex, the amplitude of the S wave decreases In the lead from the left sternal border normally the ratio of R S is at least 1 10 Reference was made earlier to the fact that an "abnormal" (inverted) T wave is occasionally found in normal individuals This occurrence is much rarer in a lead from the cardiac apex than in one from the left sternal border

The following observation suggests the use of caution just as in leads from the extremities, an alteration of the position of the heart during quiet respiration can decidedly influence the initial and terminal deflections In Fig 79 two chest leads from the left leg and the left sternal border are reproduced, they were obtained from healthy individuals In the upper tracing the T waves periodically become higher and lower with respiration and the R waves also vary in height In the lower tracing the variation of form of the initial deflection is much greater, the T wave becomes even inverted during inspiration The standard leads from these patients showed no abnormality We have frequently observed

method, the exact location of the cardiac apical region in the recumbent patient by means of fluoroscopy, likewise cannot be accomplished in actual practice

The *American Heart Association* and the Cardiac Society of Great Britain and Ireland recommend *that those who employ a* single præcordial lead should place the præcordial electrode on the extreme outer border of the apex beat or just outside the left border of cardiac dullness in the fifth intercostal space If percussion of the heart is unsatisfactory the electrode should be placed just outside the midclavicular line This lead should be called Lead IV R when it is paired with an electrode on the right arm or Lead IV F when the other electrode is placed upon the left leg A circular electrode *between 2–3 cm in diameter is recommended*

In the light of the present status of our knowledge it seems best to record leads from *both* the cardiac apex and from the left lower parasternal region *One* lead does not suffice The lead from the cardiac apex has all the disadvantages detailed above one from the lower left sternal border may in cases of coronary thrombosis produce a normal electrocardiogram when the apical lead shows distinct alterations (Fig 80 Case 4)

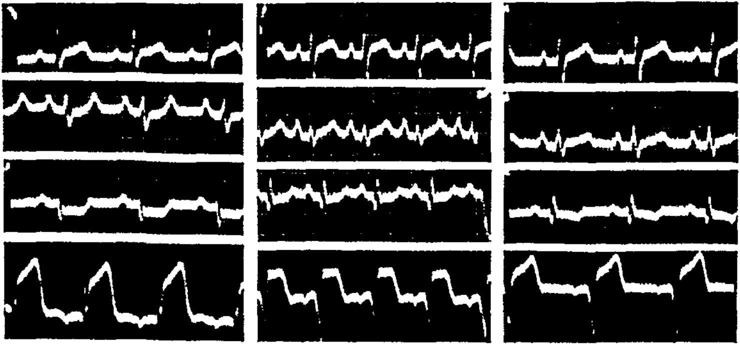
In Fig 80 a series of tracings from eight cases are reproduced below each other *Under the three leads from the extremities there is* a chest lead taken from the right arm to the left sternal border and lowermost, a chest lead from the right arm to the cardiac apex All the electrocardiograms were recorded after careful standardization

In the first tracing at the left (Case 1) there is a normal electrocardiogram In the chest leads we see distinctly how the R wave in the lead from the cardiac apex and the S wave from the lead at the left sternal border are larger At the cardiac apex the T wave is higher In Case 2 *the tracing of a simple levocardiogram* is reproduced Here also, in the leads from the extremities as well as from the chest there is no evidence of myocardial disease Distinct U waves are visible in the chest leads In Case 3 a Q wave an upwardly convex S–T segment and the cove plain T wave indicate the presence of cardiac infarction in the anterior wall The electrocardiogram was recorded from a patient who a few days before had actually experienced an attack of angina pectoris *lasting several hours In the chest leads the* R wave is absent, the S–T segment is markedly elevated and passes with an arc into the inverted T wave In Case 4, a forty one year old man, there is a levocardiogram, the only striking feature is the small negative after deflection following the R wave in Lead I Physical examination strongly suggested a

in children Case 6 was a fifty one year old patient who had suffered from many contusions and a blunt injury in the region of the chest The electrocardiogram recorded from the extremities shows a low T wave in Lead I and negative T waves in Leads II and III The chest lead from the left lower sternal border is normal the lead from the cardiac apex shows a bifid T wave The electrocardiogram of Case 7 shows in all leads from the extremities as well as in the chest leads very distinct alterations which result from digitalis therapy The tracing was obtained from a thirty six year old decompensated patient with œdema and ascites The typical slow descent of the S–T segment to below the isoelectric line and its rapid rise is distinct Tracing 8 was obtained from a seven year old child who was suffering from thyrotoxicosis Even in the leads from the extremities split T waves are visible in Lead II Distinct bifid T waves are visible in both chest leads Since split and bifid T waves may be found in normal children this electrocardiogram is not definitely abnormal

As mentioned earlier the absence of the R wave in the apical or both chest leads is regarded as pathological It is absent in *cases of coronary infarction with occlusion of the descending branch of the left coronary artery (Fig 80 Cases 3 and 4) But the R wave may occasionally be absent also in hypertension in lesions of the aortic valves with an excessive strain on the left ventricle* less often it is absent in cases of mitral valve lesions with a predominance of regurgitation and in simple coronary sclerosis without thrombosis of an artery The *S T segment* normally is somewhat elevated but in coronary occlusion (Fig 80 Cases 3 and 4) and in hypertrophy of the left side of the heart it may become displaced much higher Reference has earlier been made to the fact that likewise a *negative T wave* may occur normally and this finding alone does not justify a report that the heart is affected

In Fig 81 the electrocardiogram is that of a patient who about eight hours earlier had had a very severe pain behind the sternum suggesting a coronary thrombosis In Fig 81a only a slight elevation of the S-T segment in Lead I and a distinct depression of it in Lead III is visible The chest lead shows a typical high take off Three days later (Fig 81b) *there was a deeper S wave in Lead I a higher T wave in Lead III and the alterations of the final deflection are less distinct In the chest lead the R wave is absent* the S–T *segment and the T wave are displaced high above the O line* There is sinus tachycardia Three days later (Fig 81c) the electro cardiogram from the extremities was normal However the absence



a b c

Fig. [illegible] Limb leads and the lead from the right arm to the cardiac apex (lowermost tracing) in a case of coronary thrombosis

of the R wave in the chest lead and the high S-T segment would—in conjunction with the history—permit the diagnosis

In cases of *infarction of the anterior wall* the chest electrocardiogram is altered with great regularity at times even when no changes have yet occurred in the leads from the extremities The absence of the R wave is the most constant sign particularly in the late stages Occasionally a small Q wave precedes the R wave while this seems to be pathological in the lead from the cardiac apex it seems to occur normally in the lead from the left lower sternal border The high take off or more frequently the elevation of the S-T segment soon vanishes and inverted T waves are not regularly present In an occlusion of the right descending coronary artery (posterior wall infarction) the chest lead usually is normal or shows alterations which are not characteristic

Very large T waves in the chest lead have been described by Wood and Wolferth as a sign of coronary infarction Often they may be obtained only from circumscribed areas of the præcordium However a diagnosis should not be made from them alone since in hyperexcitable cardiac action (irritable heart) very high T waves may also be found in leads from the extremities

As in the leads from the extremities slight splintering of the initial deflections is found normally An M or W form of the initial complex is often noted in mitral valve lesions Small excursions (low voltage) also occur in the chest leads but much less often than in leads from the extremities The reasons for this difference are not clear at present

Naturally digitalis alters the chest leads as well usually in the form of a depression of the S T segment (Fig 80 Case 7)

It is undoubtedly true that in the most diverse myocardial diseases alterations in the chest leads at times may appear earlier than in the leads from the extremities Accordingly the chest leads supplement the standard leads from the extremities *But the value of chest leads is often exaggerated* Many problems are still unsettled and only a short time has elapsed since the most sweeping deductions were drawn from alterations which to day are regarded as normal variations Coronary thrombosis was often assumed to exist on the basis of an abnormal ' chest lead *But fortunately serious* criticism was soon directed against these diagnostic errors (Roth) Through incessant investigation an increasing number of variations of the normal electrocardiogram are becoming known and even if medicine is still far removed from a complete knowledge of all the *necessary rules and deviations* much has been learned in recent

years To day it is known that many reports were exaggerated which described "typical" abnormal chest lead electrocardiograms, despite normal leads from the extremities Upon exact inspection most of the leads from the extremities also disclose distinct alterations, and the chest lead electrocardiograms were not less atypical

In a majority of cases, especially in the analysis of disturbances of rhythm, the classical electrocardiogram with leads from the extremities suffices When the electrocardiogram of the chest lead alone is abnormal, a diagnosis of a cardiac disease should rarely be made exclusively on this basis, but the discovery of an abnormal chest lead is sufficient to warrant great care in the observation of the patient and repetition of the examination Concerning the areas most suitable for chest leads, the question is still open

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DISTURBANCES OF STIMULUS FORMATION AND CONDUCTION

The fundamental condition for normal cardiac action is the regular formation of stimuli and their normal spread over the heart (stimulus conduction) followed by a powerful contraction. Disturbances of cardiac activity will appear when these basic functions are abnormal. Disturbances of contractility, as was emphasized earlier in this book, cannot be recognized by the electrocardiograph. It is impossible to differentiate the electrocardiogram of a compensated heart from a tracing obtained from a decompensated patient. But disturbances of stimulus formation and conduction can be detected by the electrocardiogram better and sooner than by any other method of investigation.

As a matter of fact most arrhythmias had been discovered before the electrocardiograph was invented; thus the first description of periodically dropped beats in man by means of an analysis of the radial pulse tracing (Wenckebach) will always remain a great achievement. But in this case the electrocardiogram makes the diagnosis possible at a mere glance, whereas formerly it could be obtained only after a painstaking and time consuming analysis of laboriously recorded tracings of the radial and venous pulse as well as of the impulse at the apex of the heart.

Disturbances of stimulus formation present a variety of pictures; *indeed there may be simply an increase or decrease of normal* stimulus formation or automatism (sinus bradycardia, sinus tachycardia) or new automatic centres may appear (auriculo-ventricular (A V) rhythm, parasystole, interference of two rhythms) or a pathological entirely abnormal type of stimulus formation may occur as in extrasystole, paroxysmal tachycardia, flutter and fibrillation.

The disturbances of conduction as far as they are located below the bifurcation of the bundle, that is in the two branches, were discussed in the preceding sections of this book. The electrocardiogram is indispensable for the recognition of these disturbances of intraventricular conduction. If a myocardial disease injures the ramifications of the conduction system, the spread of excitation and therefore the form of the ventricular complex is altered.

In later chapters of this book the disturbances of conduction

which originate in the A–V system above its bifurcation (A–V conduction disturbances) are discussed then the conduction disturbances in the auricle (disturbances of intra auricular conduction) and the abnormalities of conduction between the sinus node and the auricle (sino auricular conduction disturbances) find consideration

EXTRASYSTOLES

Introduction

Every specific fibre of the heart can form stimuli without any external influence, "from itself alone" it can become *automatically* active The same situation holds for the specific fibres of the *auricles (sinus node with its junctional fibres auricular part of the* A–V node with junctional fibres) and for the specific fibres of the ventricle (ventricular part of the A–V node, bundle of His bundle branches with arborizations, Purkinje fibres)

If in an experiment, the sinus node is divided into the smallest just visible parts each part shows rhythmic contractions as can be easily *demonstrated macroscopically*, *they form stimuli*" (v Skramlik) Even the smallest fragments removed from the branches of the atrio *ventricular conduction system exhibit movements as soon as they are* placed under suitable conditions (proper nutrient solutions, warming to 37° C introduction of oxygen) A strip of muscle removed from the auricle or ventricle also shows rhythmic contractions, if it remains in contact with the specific fibres If small parts of the ramifications of the A–V system (incorrectly called Purkinje fibres) are removed from the heart two to three days after death and observed under the microscope, they still exhibit movement (Ishihara and Pick) The fibres of the specific tissue constitute the ultimum moriens in the heart (Pick)

Whether or not rhythmic stimuli are formed in the common muscle is dubious since all investigations up to the present time have failed to prove it Even when stimulus formation in the specific tissue is increased to the utmost by poisons such as barium, no stimulus formation is found in the common muscle (Scherf) Under the influence of chemical substances (concentrated salts) one may see, transiently, rhythmic contractions in the common muscle, but it is not proven whether or not *a continuous formation of rhythmic stimuli is possible in the heart outside* of the specific fibres However unpublished experiments show that the *application of concentrated solutions of salts or of barium to the* epicardium provoke extrasystoles and tachycardia Accordingly a final *decision has not as yet been reached*

If every specific fibre possesses *the property of forming stimuli*, it must be explained why normally they are silent and inactive, why

the normal cardiac stimulus develops *only* in the fibres of the sinus node. Two fundamental laws of the heart explain these facts —

1. The nearer the specific fibres are located to the apex, the less is the development of their automatism and the slower they work. The fibres of the sinus node (the primary centres) have the most highly developed automatism, the fibres of the A V node (secondary centres) have a slower automatism, the Purkinje fibres in the periphery (tertiary centres) work the slowest.

2. If a stimulus formed in the head of the sinus node spreads normally over the heart, it breaks through every other more slowly working centre[1] and destroys the stimulus material accumulating there. Everywhere the formation of stimuli must begin anew. But since stimuli are produced most rapidly in the head of the sinus node they always appear earlier than stimuli in the other centres and prevent the latter from emerging. Under normal conditions, therefore, the following rule holds (for conduction): *that centre controls the heart which forms stimuli at the highest rate.*

If the head of the sinus node becomes incapacitated through disease, a lower area of the sinus node that has remained intact assumes control. If the entire sinus node no longer forms stimuli, the secondary centres in the A V node step in. A similar event occurs when the secondary centres fail and the tertiary centres assume control. All the deeper centres are, to some extent, on the alert, and immediately appear in the breech when higher centres cease to work or act too slowly, or when owing to an interruption of conduction the stimuli formed by them are not transmitted.

At times a specific fibre in the heart can be altered in some way (the finer details being still unknown) so that the preceding excitation induces it to form a stimulus by itself; this stimulus, if above threshold, may produce a new cardiac contraction. These abnormal premature contractions which disturb the normal rhythm are called *extrasystoles*. They do not owe their origin to an automatic (intrinsic) formation of an impulse but to an abnormal pathological stimulus. Since the extrasystoles are released and produced by the beat preceding them they always appear at a shorter or longer interval from the preceding beat; they are always bound or coupled to it. In every case this time relation is remarkably constant (fixed coupling).

Extrasystoles should always be suspected when premature beats are

[1] Whether a "centre" consists of *one* specific fibre or a group of fibres has not been decided.

found in the electrocardiogram and when they exhibit a constant, usually short, coupling to another beat

Since there are specific fibres in the auricles and ventricles extrasystoles can be divided into two main groups auricular and ventricular extrasystoles (*Extrasystoles which develop in the A–V node are discussed later, p 317*)

Although in animal experiments extrasystoles had earlier been observed, they were described for the first time in man by Wenckebach and Cushny (1899)

Ventricular Extrasystoles

The discussion may be introduced by a consideration of *ventricular extrasystoles* and by an analysis of the disturbance of the rhythm which they produce

The drawing in Fig 82 shows at the left the course of three normal stimuli over the heart The stimuli originate in the sinus node and are conducted from there to the auricle and then to the ventricle Shortly after the third normal beat, a ventricular extrasystole appears It is a rule that ventricular extrasystoles in man are conducted back to the auricle *only under very exceptional circumstances This occurs (in con* trast to the dog) so rarely that only a few cases of this kind have been described in medical literature (Samet) To be sure the bundle of His readily conducts a stimulus backward toward the auricle but the A–V node, which is a rather poor conductor even in the normal direction, blocks almost all retrograde stimuli

Fig 82 (Schematic) Disturbance of rhythm caused by a ventricular extrasystole

Since ventricular extrasystoles are not conducted to the auricle in a retrograde manner naturally they do not reach the sinus node and thus do not disturb stimulus formation there *In other words ventricular extrasystoles produce a disturbance of rhythm which is limited to the ventricle* The activity of the sinus node and of the auricle proceeds undisturbed, just as if no ventricular extrasystole had occurred The sinus stimulus 4 in the drawing however cannot reach the ventricle since on its journey, *in the A–V system*, it encounters tissue which is still refractory and which has not recovered , the stimulus remains ineffective , it is blocked and the fourth ventricular systole does not follow The fifth and all subsequent stimuli reach the ventricle in a normal way

The disturbance of rhythm caused by a ventricular extrasystole consists, therefore, simply in the appearance of an abnormal premature ventricular beat, and results in the omission of a normal contraction Since the first beat after the extrasystole occurs at the same time at which it would have appeared had there been no extrasystole, the interval between the last normal beat before, and the first normal beat after, the extrasystole (between beats 3 and 5 of Fig 82) is just as long as two normal periods This fact was discovered by Engelmann on frog hearts and was confirmed by Wenckebach in the human heart, it is called *the law of maintenance of the stimulus period* The pause between the extrasystole and the next normal contraction is called *compensatory* because it supplements the shorter interval preceding the extrasystole to the extent that the two intervals together are as long as two normal periods

Formerly as long as only the radial and venous pulse curves were recorded, considerable importance was attached to measuring the length of the post extrasystolic interval But, at present, when the employment of the electrocardiogram has become widespread the diagnosis can be made at a glance so that the length of the post extrasystolic interval is no longer important Since respiratory arrhythmia is so common in healthy individuals the individual normal periods vary in length so that no comparable values can be obtained

Corresponding to the appearance of a premature ventricular contraction, we see a premature ventricular complex in the electrocardiogram, no P wave precedes it, since the stimulus arises in the ventricle and is not conducted back to the auricle

The ventricular complex of the extrasystole varies in shape according to the site of its origin If, for example the origin of the extrasystole is located above the site of bifurcation of the bundle, the excitation spreads normally over the two ventricles and the ventricular complex appears identical to any other normal complex of the same case But extrasystoles of this type are rare Experience shows that *the centres endowed with the strongest automatism (sinus node A–V node bundle of His) are the rarest sites of origin for extrasystoles* On the contrary, the fibres lying more deeply (the nodal junctional fibres in the auricle, the finer and finest ramifications of the A–V system in the ventricle), are very frequently the point of origin for extrasystoles The further one passes along the A–V system toward the cardiac apex, the less the tendency to formation of automatic stimuli and the greater the tendency to the formation of extrasystoles becomes Thus it happens that the vast majority

of ventricular extrasystoles arise far below the site of division of the bundle

Let us assume that an extrasystole develops in the left ventricle (at x, indicated in the drawing of Fig 83) In this case, since the stimulus originates in the left ventricle, naturally this structure is activated first and the right ventricle is excited subsequently The progress of excitation resembles that which is found in right bundle branch block An electrocardiogram from such a case shows a levo cardiogram, widening and splintering of the QRS complex, and a terminal deflection which is directed opposite to the initial complex *If one removed from a tracing a single ventricular complex of this type, it would be impossible to determine whether an extrasystole or a bundle branch block existed* The presence in a tracing of a single premature beat not preceded by a P wave provides the diagnosis

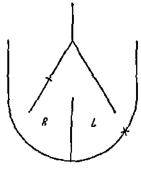

Fig 83 Schematic representation of the spread of excitation of a ventricular extrasystole

Fig 84 shows an electrocardiogram obtained during an experiment It was obtained from a dog whose thorax and pericardium

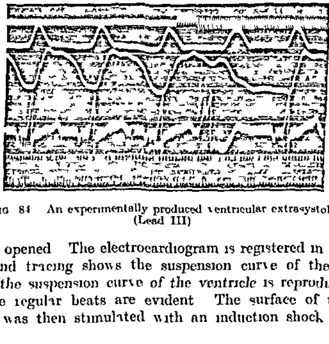

Fig 84 An experimentally produced ventricular extrasystole (Lead III)

had been opened The electrocardiogram is registered in Lead III The second tracing shows the suspension curve of the auricle, *below it the suspension curve of the ventricle is reproduced* At first some regular beats are evident The surface of the right ventricle was then stimulated with an induction shock As the

stimulus signal (shown in the top tracing) shows the break shock produced a ventricular extrasystole The electrocardiogram reveals all the features mentioned above A premature abnormal ventricular complex appears it is not preceded by a P wave The extrasystole shows widening and splintering of the QRS complex, its terminal deflection is markedly depressed below the isoelectric line and directed opposite to the initial complex From the suspension curve of the auricle it is evident that the auricle continues to contract rhythmically without any disturbance whatsoever Likewise

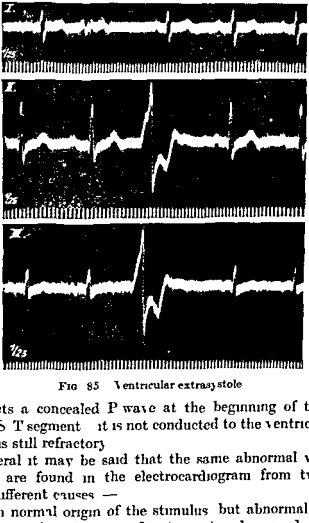

FIG 85 Ventricular extrasystole

one suspects a concealed P wave at the beginning of the deeply depressed S T segment it is not conducted to the ventricle because the latter is still refractory

In general it may be said that the same abnormal ventricular complexes are found in the electrocardiogram from two fundamentally different causes —

1 With normal origin of the stimulus but abnormal spread of excitation (in disturbances of intraventricular conduction for example in bundle branch block)

2 With abnormal origin of the stimulus (for example, extra systole or automatic "idioventricular" beats)

Fig 85 shows a regular normal "sinus rhythm" except for a disturbance caused by a premature abnormal ventricular contraction A single abnormal QRS complex with an abnormal terminal deflection appears in all leads The premature abnormal QRS complex in Lead I is very small and almost isoelectric The post extrasystolic pause is compensatory since measurement shows that the interval from the last normal beat before the premature contraction to the first beat after it is just as long as two normal periods

The ventricular complex of the extrasystole is abnormal not because the myocardium or the conduction system is damaged but is altered because the extrasystole originates in an abnormal place and spreads abnormally over the ventricle

In spite of the extrasystole the auricle contracts without disturbance Measurement shows that a P wave must be concealed in

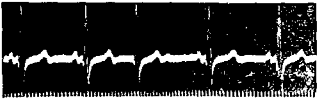

Fig 86 An extrasystole which arises from above the site of bifurcation of the bundle

the ventricular complex of the extrasystole, because it is so small, the P wave is usually invisible

In Fig 86 two normal beats with well formed P waves and normal initial complexes can first be seen then a premature ventricular complex, not preceded by a P wave is noted However this premature contraction has exactly the same shape as the other sinus beats, therefore it must be assumed that a ventricular extrasystole is present which originated above the bifurcation of the bundle of His For this reason its spread in the ventricles is the same as that of sinus beats With more exact inspection one discovers that the R wave of the extrasystole is somewhat higher than those of the normal beats this occurs because the normal P wave and the R wave of the extrasystole coincide The potentials of the auricle and ventricle summate and the R wave is higher

The endeavour to establish the site of origin of ventricular extrasystoles was initiated by Einthoven According to him a distinction can be made between right and left ventricular extra

systoles by examination of Lead I If in Lead I the initial deflection of the extrasystole is directed upward the extrasystole originates in the right ventricle if it is directed downward the extrasystole arises from the left ventricle Lead III (at times Lead II) makes possible the differentiation between basal and apical extrasystoles When the stimulus originates near the cardiac apex the initial complex is directed downward in Lead III if the extrasystole arises near the base of the heart the initial deflection is directed upward in this lead More recent investigations by Storm on hearts *in situ* of monkeys are also in favour of this interpretation According to him it is entirely immaterial in respect to the direction of the deflection in Lead I whether the extrasystole arises in the anterior or posterior aspect of the ventricles whether above or below for in this lead the initial complex always is directed upward in right ventricular extrasystoles and downward in left ventricular extrasystoles If the extrasystole originates in the groove between the two ventricles in the region of the descending ramus of the left coronary artery the ventricular complex of the extrasystole shows a mixed form

Investigations have also been conducted on the human heart which for some reason has been exposed they confirm these statements but also disclose many exceptions to this rule This is understandable because the position of the heart can also decisively influence the direction of the deflection

With these reservations it may be said that the extrasystoles in Fig 85 originate from the intraventricular groove near the base of the heart on the other hand those of Fig 87 arise from the apical region of the right ventricle

However it is impossible at the present time to state with assurance the point of origin of a ventricular extrasystole moreover a statement of this kind has relatively little diagnostic import Nevertheless these experimental findings possess great significance from a theoretical standpoint because they tend to support the new nomenclature of bundle branch block they furnish evidence to corroborate the idea that when the initial deflection is directed upward in Lead I the right ventricle is activated first and when the initial deflection is directed downward in Lead I activation is initiated in the left ventricle (Wilson and co workers)

In Fig 87 a ventricular extrasystole is seen in each lead Its initial deflection shows a high R wave in Lead I and a deep S wave in Lead III In all probability it is an extrasystole originating in the right ventricle

The electrocardiogram of the sinus (normal) beats of this case is not normal since the T waves are very low. The tracing was obtained from a patient who suffered from aortitis with coronary stenosis, when the heart was embarrassed after stair climbing, the electrocardiogram became very abnormal.

It was mentioned earlier that in ventricular extrasystoles the activity of the auricles is not disturbed. Stimulus 4 of Fig. 82, however, cannot reach the ventricle, because the latter is still *refractory after the* ventricular extrasystole. The blocked P wave is usually not visible since it is hidden in the ventricular complex of the extrasystole. At times, as in Fig. 84, it is visible between the QRS *complex and the* T wave of the extrasystole. But if the heart

Fig. 85. Ventricular extrasystoles and signs of myocardial injury in the electrocardiogram (abnormal T wave in Lead II).

beats slowly and the extrasystole occurs very early in diastole, then the auricular stimulus (No. 4), which is otherwise blocked, may *appear at a time when the refractory stage of the conduction system* and of the ventricular muscle has already ended, in this case the stimulus may be conducted to the ventricle and is not blocked on the way. If this happens the long post extrasystolic pause is not found and the interval between the last normal beat *before* and the first *after* the extrasystole tends to equal *one* rather than two normal periods. Since in such case, the ventricular extrasystole is inserted between two normal beats, without disturbing the normal rhythm it is called an *interpolated extrasystole*.

Since the normal beat after the interpolated extrasystole is often conducted somewhat slowly to the ventricle because the conduction

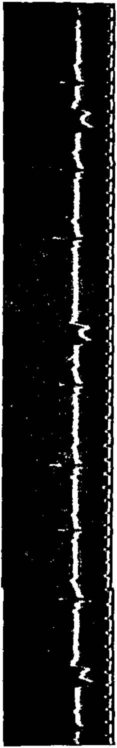

Fig 88 Three extrasystoles, the third of which is interpolated

system has still not completely recovered from the extrasystole, this interval is usually somewhat longer than a normal period and the following one is just as much shorter

In Fig 88 at the beginning of the tracing a ventricular extrasystole appears after a normal contraction, the electro cardiogram is that of a patient with respiratory arrhythmia The normal P wave (corresponding to No 4 of the drawing in Fig 82) is visible in the S-T segment between the initial complex and the T wave But the P wave is not followed by a ventricular complex, because the stimulus on its way to the ventricle encounters tissue which has still not recovered, and is therefore incapable of conduction For this reason a long post-extrasystolic pause follows After four normal beats a ventricular extrasystole again appears The P wave of the next normal stimulus coincides with the T wave of the extrasystole, and likewise is not conducted to the ventricle After two more normal beats a third extrasystole occurs The P wave of the next normal stimulus, which ordinarily is hidden in the QRS complex or the T wave of the extrasystole this time appears shortly *after* the T wave of the extrasystole since owing to the respiratory arrhythmia the P–P interval is somewhat longer This stimulus which appears late in diastole, finds the bundle capable of con duction and reaches the ventricle with some delay (the P–R interval is prolonged), thus the third extrasystole is interpolated

After this extrasystole no long post-extrasystolic pause follows and the interval between the last normal beat before and the first after this extrasystole is somewhat longer than a normal period This slight delay is due to the fact that the post-extra

systolic normal beat is conducted slowly to the ventricle. The *subsequent pause is therefore shortened by the corresponding extent.*

Interpolated extrasystoles have no different significance than non-interpolated ones. As a matter of fact their appearance merely depends upon whether or not the time relations permit the conduction of the first auricular stimulus after the extrasystole. Only when a great many interpolated extrasystoles appear—an unusual occurrence—are they unpleasant since they may double the heart rate and the patient may have, for example, as many as 160 beats per minute in place of 80.

In Fig. 89 a series of interpolated ventricular extrasystoles appear after two normal beats; in this instance they are inserted in succession between two normal beats. As a result the heart rate is doubled from 62 to 124 beats per minute. *The interpolation of extrasystoles is assisted by the existing bradycardia. The normal* beats between the extrasystoles show slight differences in the

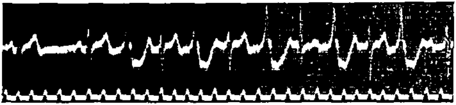

Fig. 89. A series of interpolated extrasystoles.

form of the QRS complexes and the T waves. This happens because the preceding diastoles are greatly shortened by the pre-*ceding interpolated extrasystoles and the paths of conduction in the* ventricles have still not completely recovered when the normal beat appears; the normal contraction then spreads abnormally in the ventricles and an abnormal electrocardiogram results.

Owing to the shortness of the preceding diastole the normal beat after an interpolated extrasystole may not only be conducted slowly in the bundle of His (the conduction system above the bifurcation of the bundle being still fatigued—atrioventricular conduction disturbance) but *also show an abnormal ventricular* complex (the conduction path below the bifurcation having not yet recovered—disturbance of intraventricular conduction). These variants are devoid of practical significance.

After the ordinary non-interpolated ventricular extrasystole only rarely does one find an abnormal shape of the post-extrasystolic normal beat since the long post-extrasystolic interval makes possible

sufficient recovery of the conduction paths The form of the T i occasionally changes in the first post-extrasystolic beat

In accordance with the preceding discussion ventricular e systoles which originate in the same place and in the same abno centre spread over the heart in the same way and always pro the same electrocardiogram Moreover if one has the opportu of observing extrasystoles in the same patient for years their elec cardiogram shows continually the same shape , the smallest n and the finest splintering remains the same It is very rare to extrasystoles of two shapes in the healthy individual , in this only these and no intermediate forms occur

If an organic disease of the heart is present *several* spe fibres may become affected in such a way that they form abnor stimuli , owing to the multiple sites of origin extrasystoles w are polytopic (multifocal) show various forms In this instance sees in the electrocardiogram a continual variation in notch forma and splintering as well as alterations in the height and direc of the waves Disturbances of intraventricular conduction ca by the prematurity of the extrasystoles also participate in produ the varying forms of extrasystoles in organic heart diseases premature contraction appears so early in diastole that the t has still not fully recovered from the preceding normal excitat for this reason the extrasystole does not always spread thro the ventricle in the same way Since the irritability of the h varies especially that of the pathological heart, the spread of extrasystolic activation is not always the same

In myocarditis, in the heart muscle injuries of diphtheria coronary sclerosis and myomalacias multiform extrasystoles frequent Often they constitute an early symptom of an orga disease of the heart muscle and they have the same significa as an inverted T wave or widening of the initial deflection

Fig 90 was obtained from a sixty four year-old untreated pat who came to the clinic because of nocturnal attacks of card asthma Physical and X ray examination disclosed a heart nor in size and shape the heart sounds were pure and the blood press was normal The electrocardiogram (Lead II) shows a sligh inverted T wave and the assumption of a myocardial injury seen probable

However the electrocardiogram showed in addition four v tricular extrasystoles all of which exhibited different for Tracings many feet in length had to be recorded before two ext systoles were found which were identical

To be sure one may not assume that the absence of a continuous change in the form of the extrasystoles excludes an organic basis Even *when the extrasystoles have an identical shape a myocardial* affection *may* exist In this instance the lesion has awakened only *one* centre of abnormal stimulus formation Only the opposite rule holds *ventricular extrasystoles of diverse shapes favour the existence of organic cardiac disease*

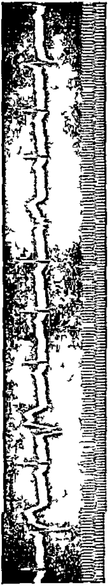

Fig 90 Var form ventricular extrasystoles in coronary sclerosis

Extrasystoles *may appear during digitalis* therapy *Digitalis extrasystoles* constitute one of the most important signs of so called d i g i t a l i s intoxication Clinical experience shows that digitalization alone does not suffice for producing extrasystoles Clinicians have for a long time been aware that in many cases *despite the administration of digitalis for* months no extrasystoles appear whereas in other cases even a few doses may suffice to produce a continuous bigeminy (Huchard Edens) In animal experimentation regular digitalis bigeminy can never be obtained by digitalis poisoning *alone* (Kobacher and Scherf)

On the basis of our present knowledge it may be said that in addition to the effect of digitalis other conditions must be present in the heart for extrasystoles to appear these conditions are still uninvestigated and may vary from case to case Observation at the bedside shows that the greater the damage of the heart muscle the more readily do extrasystoles appear after digitalis (they are always ventricular extrasystoles) *accordingly they represent an unfavourable prognostic sign* At all events digitalis extrasystoles imply that the heart is not healthy Hypertrophy and dilatation of the heart alone do *not* augment the tendency of digitalis extrasystoles to appear

Since digitalis acts upon the entire heart upon *all* its centres and since digitalis extrasystoles appear only in damaged hearts

they also show varying forms (Scherf) This rule also has no exceptions In some rare instances at first there is only a change in the amplitude of the waves, in the appearance of varying thickness and notching (slurring) of the limbs of the waves These changes increase as digitalis is continued But in most cases of digitalis extrasystoles even soon after their appearance, one finds the change in the form of the ventricular complex of the extrasystole so marked that often long tracings must be registered in order to find two beats which appear identical

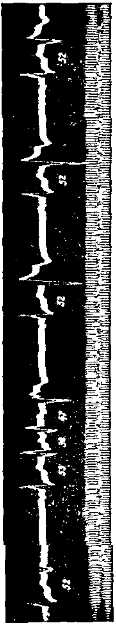

Fig 91 Ventricular extrasystoles of varying forms during digitalis therapy

Fig 91 was recorded in a patient with a severely decompensated mitral valve lesion he had been under treatment with digitalis for a long time One finds negative P waves slight notching and thickening of the R waves as well as a definite depression of the S-T segment and a biphasic T wave (Lead III) Moreover, ventricular extrasystoles are present, at one place multiple extrasystoles appear The extrasystoles are so varied in form that none are alike

In other words, if one sees ventricular extrasystoles of varying appearance in the electrocardiogram, myocardial disease is always present, very often this disease alone is not responsible for the extrasystoles, superimposed digitalis therapy may have been provocative

While digitalis extrasystoles appear in one individual after a few doses of digitalis in another patient they develop only after the administration of large doses They may vanish within a few days, but sometimes they persist for two or three weeks after interrupting the treatment

It is important to know whether or not digitalis therapy should be continued when extrasystoles of the type just described have appeared This would never be permissible if bigeminy were

exclusively a sign of digitalis intoxication But as we have pointed out extrasystoles are merely a symptom accompanying the action of digitalis and their appearance depends to a great extent upon the state of the heart muscle at the time Since an injury of the myocardium appearing during the course of decompensation as the result of an accumulation of metabolic products from oxygen deficiency etc contributes to the appearance of extrasystoles it is quite possible that extrasystoles may appear after small doses of digitalis but vanish after larger amounts their disappearance being the result of the removal of the decompensation and the consequent elimination of one of the precipitating causes of extrasystoles

For this reason when extrasystoles appear during the digitalis treatment of an incompletely compensated patient it is well to continue treatment with small doses of digitalis (perhaps 0 15 gm

FIG 92 Numerous ventricular extrasystoles of various forms after digitalis strophanthin

(1½ grs) daily) *in this instance the patient should be examined* daily and three groups of cases may be distinguished according to their subsequent development

1 *In rare cases the digitalis bigeminy does not change and one* succeeds in introducing an adequate amount of digitalis into the heart

2 In many cases the extrasystoles which appeared after small *doses of digitalis disappear with continuation of the therapy and do* not return At times a reduction of the amount of digitalis suffices to permit the therapy to be continued

3 In the majority of cases however the number of extrasystoles increases and if therapy is continued trigeminy appears in place of bigeminy then therapy should be interrupted at once in order to avoid a dangerous ventricular tachycardia

Since one can never predict how a case of digitalis bigeminy will react to the further administration of digitalis and whether or not it belongs to that group in which the extrasystoles will immediately multiply if therapy is continued the injection of strophanthin is contraindicated in the presence of a digitalis bigeminy

Fig 92 was obtained from a severely decompensated case of

emphysema During digitalis treatment which failed to yield any improvement bigeminy appeared Since the physician in charge did not know that strophanthin is contraindicated in the presence of digitalis bigeminy he gave an intravenous injection of ¼ mg (1/250th gr) of strophanthin Fifteen minutes later a very fast arrhythmia appeared After three normal beats (after the pause) seven ventricular extrasystoles appear all are different in shape A few minutes later the patient died in an attack of ventricular fibrillation Auricular fibrillation is also evident in the electrocardiogram

The same rules also hold for the extrasystoles produced by squill adonis helleborus and their derivatives

The same kind of extrasystoles appear during chloroform

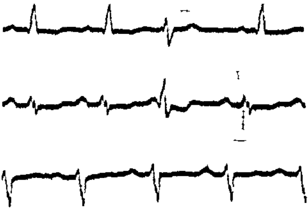

FIG 93 Signs of myocardial disease and belatedly appearing ventricular extrasystoles.

anaesthesia The serious cardiac symptoms and the tragic instances of death in chloroform anaesthesia were explained with the aid of the electrocardiogram (Levy) It disclosed at first isolated and then variform ventricular extrasystoles in increasing numbers if the anaesthesia was continued the irregularity was converted into ventricular fibrillation

In the vast majority of cases the extrasystole is bound firmly to the preceding beat which releases it in some way it is coupled This coupling is usually exact to a hundredth of a second Great variation in the coupling suggests the presence of an interference of two rhythms or parasystole (p 321) Under these circumstances one must record tracings several feet in length in order to obtain an

exact analysis of the situation The extrasystoles may come very early so that their ventricular complexes begin on the descending limb of the T waves of the preceding beats that is at a time when the absolute refractory period has just ended But extrasystoles may also occur so late in diastole, that in bradycardia one may find a coupling of 100–200 hundredths of a second

At times extrasystoles appear so late in diastole that the normal P wave precedes them although with a shortened interval Thus Fig 93 shows ventricular extrasystoles appearing in a case of severe coronary sclerosis There exists a levocardiogram and the initial complexes are plump and notched The width of the initial deflections amounts to 0 10 second The S–T segments in Leads I and II are below the isoelectric line and there are no T waves In Leads I and II a ventricular extrasystole appears after every two normal beats It comes so late that it is preceded by a (normal) P wave If the stimulus producing the ventricular extrasystole had arrived a few hundredths of a second later no extrasystole would have occurred since the normal stimulus would have preceded it

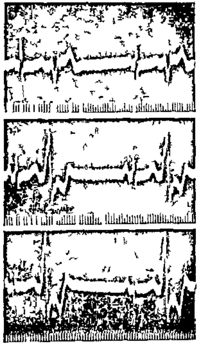

Fig 94 Ventricular bigeminy

Extrasystoles may be isolated events or they may follow every 20th, 12th, 5th beat, etc , they may also come after every normal beat (bigeminy)

In Fig 94 a ventricular extrasystole appears after every normal beat, so that ventricular bigeminy is present The electrocardiogram of the sinus beat is normal, the small initial deflection in Lead III may be found in the healthy individual with the corresponding direction of the cardiac axis (see p 28)

A continuous ventricular bigeminy is also present in Fig 95 But in this instance the electrocardiogram of the sinus beats is abnormal The form of the P waves undergoes continual change in each lead in Lead II abnormal inverted P waves are evident (disturbances of intra auricular conduction, see p 295), neverthe

less the conduction time is normal. The initial deflections in Leads I and III are notched and 0.09 second in width. In Lead I the T waves are slightly inverted; in Leads II and III a deep inversion of the T waves is evident. The tracing is that of a patient with a rheumatic insufficiency of the aortic valve before treatment.

FIG. 95. Ventricular bigeminy; signs of myocardial damage in the electrocardiogram and disturbance of intraventricular conduction.

If two extrasystoles appear after every normal beat a trigeminy exists. Trigeminy is often wrongly assumed to be present when one extrasystole occurs after two normal beats. If many extrasystoles follow each other in direct succession they are called *multiple*

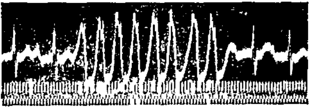

FIG. 96. Multiple ventricular extrasystoles; the sinus beats are slowly conducted.

extrasystoles. There are gradual transitions from this condition to the tachycardias.

Fig. 96 shows a group of six ventricular extrasystoles. The atrioventricular conduction time of the sinus beat is prolonged to 0.26 second (see p. 276).

Auricular Extrasystoles

If a premature auricular contraction appears, it is called an auricular extrasystole In contrast to ventricular extrasystoles they disturb the rhythm of the *entire* heart They are easily con ducted in a retrograde direction to the sinus node and disturb its activity, they are also conducted in the normal direction to the ventricles

For this reason the post extrasystolic intervals are rarely compensa tory, the *interval between the last normal beat before and the first after* the extrasystole only rarely equals two normal periods this occurs only exceptionally when the auricular extrasystole happens to occur at a definite phase of diastole

Just as a premature ventricular complex occurs in the electro cardiogram of a ventricular extrasystole a premature P wave is

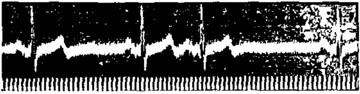

FIG 97 Auricular extrasystoles originating in the vicinity of the *head of the sinus node*

found in the record of an auricular extrasystole they exhibit different shapes depending upon the site of origin

In rare cases the extrasystole arises in a centre near the normal pacemaker that is near the head of the sinus node The spread of the excitation wave of these extrasystoles is then normal a P wave is observed which cannot be distinguished from the normal P waves of the patient Abnormal P waves are seen with abnormal location of the site of impulse formation The P waves of auricular extra systoles may be larger or smaller broader or narrower than normal P *waves, at times they are inverted and split they may be so low that they are scarcely visible*

In Fig 97 a premature wave appears after two normal sinus beats, it looks almost exactly like the P wave of the normal con traction (Lead I) consequently it is a premature abnormal stimulus which was formed in the immediate vicinity of the normal site of origin of stimuli After the premature P wave a normal ventricular complex follows since the auricular extrasystole was conducted to the ventricles in a normal manner Then a post extrasystolic pause follows as measurement shows, it is not compensatory the

interval between the last normal beat before and the first after the extrasystole is shorter than two normal periods The tracing was obtained from a person free from cardiac disease

In Fig 98 three tracings are reproduced from three different patients Lead II is shown in all tracings, the time record in the upper tracing is 1/5th of a second, in the lower two illustrations, 1/25th of a second In all tracings an auricular extrasystole follows two sinus beats In the upper and lower tracings the auricular extrasystoles have inverted P waves In the middle tracing the positive P wave of the auricular extrasystole is superimposed on the

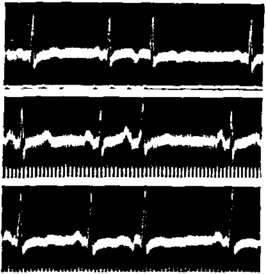

Fig 98 Three examples of auricular extrasystoles (Lead II)

T wave of the preceding normal beat, this T-wave is broader and higher than the normal T wave

The first normal P wave after the auricular extrasystole in the middle tracing is higher and sharper than the two normal P waves at the beginning of the tracing It resembles the P wave of the auricular extrasystole This phenomenon is not uncommon after an auricular extrasystole, its cause is not as yet known (Lewis, Rachmilevitz and Scherf) At times the first post extrasystolic normal stimulus appears to utilize for its spread the same path as the extrasystole

In the lower tracing the S–T segment of the normal beats is depressed, the T wave is biphasic, since this occurs in Lead II it favours the diagnosis of myocardial disease

It is impossible to determine the point of origin of auricular

extrasystoles from the appearance of the P waves Extrasystoles which arise in the region of the lower end of the *sinus node* may also have inverted P waves The lower end of the sinus node extends *in the direction of the A-V junction*

If it is true that the common muscle is not capable of forming stimuli, auricular extrasystoles on the whole can only originate in the sinus node and its junctional fibres or in the auricular part of the A-V node and its junctional fibres since no other specific fibres *are present in the auricles*

Auricular extrasystoles like ventricular *premature contractions* may be single or multiple, grouped in bigeminal or trigeminal series,

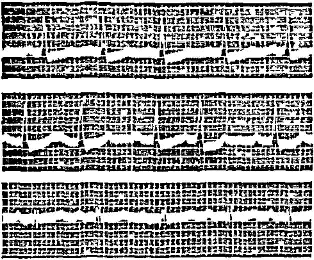

FIG 99 Auricular extrasystoles in Lead II The S-T segments are depressed in Leads I and II

or found with long or short coupling Since the refractory phase of the auricle and not of the ventricle is the factor that is decisive for their appearance, often they appear earlier than ventricular extra systoles, accordingly the P wave of the auricular premature contraction *very often is hidden in the* T wave of the preceding beat This causes a modification of the shape of the T-wave, a slight elevation or depression, slight splintering etc *If the T-wave suddenly alters its form without change in the appearance of the initial deflection, then a P wave is concealed in it* Appreciation of this basic rule prevents *overlooking* P waves which are hidden in T waves

Ventricular extrasystoles may appear very early in diastole, as a matter of fact, occasionally they are visible in the T-wave of the preceding ventricular complex and hence at the end of the absolute refractory

period in these circumstances one must assume that the stimulus producing the extrasystole is very powerful It is striking that auricular extrasystoles do not appear much earlier than ventricular extrasystoles indeed the former occur at a time when some time has elapsed since the end of the refractory period of the auricle This permits the assumption that the stimuli responsible for the auricular extrasystoles are weaker than those of ventricular premature contractions

In Fig 99 a sinus tachycardia of 115 beats per minute is present The S–T segments of the sinus beats are distinctly depressed in Leads I and II Since the patient was under the influence of digitalis this finding cannot without further information be evaluated in the sense of a myocardial disease

In Lead II the sinus rhythm is disturbed by two auricular extrasystoles One occurs after the first normal beat whose T wave appears bifid the P wave of the extrasystole being concealed in it An auricular extrasystole also appears after the following normal

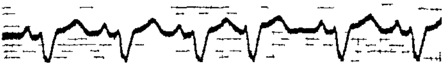

Fig 100 An auricular extrasystole with disturbance of intraventricular conduction

beat It occurs somewhat earlier in diastole so that the P wave is entirely superimposed upon the T wave One recognizes that a P wave is hidden in this T wave since the T is higher and more peaked

Fig 100 was obtained from a case with a disturbance of intraventricular conduction the common form of bundle branch block ' is present (Lead III) Here also the sinus rhythm is disturbed by an auricular extrasystole The premature P wave is concealed in the T wave of the preceding beat and is recognized by the slight alteration of the shape of the T wave

Auricular extrasystoles may like normal sinus beats be conducted through the A V system to the ventricles But if the ventricular complexes of the auricular extrasystoles are examined closely it is found that they rarely have the same appearance as the sinus beats of the same case (Figs 98 and 99) At times only a slight increase or decrease in the amplitude of the R wave the presence or absence of a Q or S wave is found In other cases however the QRS complex of the auricular extrasystole may also be widened and notched the T wave may also show corresponding changes of shape

Occasionally the ventricular complex is so altered that on superficial inspection the presence of a ventricular extrasystole might be assumed

These alterations are the result of the fact that the auricular extrasystole usually appears so prematurely in ventricular diastole that the whole of the tissue of the ventricle has not completely recovered from the conduction of the preceding normal beat Thus *it happens that a small or large branch of the conduction system is not yet able to conduct normally and the auricular extrasystole thus* spreads more or less abnormally in the ventricle *This is called aberrant conduction of the auricular extrasystole* Since at an early period in diastole, immediately after the end of the absolute refractory period even the most healthy heart has still not completely recovered, these aberrantly conducted auricular extrasystoles are found at times in the absolutely healthy heart Accordingly this *finding has no prognostic or diagnostic significance*

At a time when the auricular extrasystole is due to be conducted to the ventricles, the common path of the bundle above the bifurcation has sometimes not completely recovered from the conduction of the preceding beat In such case the auricular extrasystole is *slowly* conducted to the ventricle and the P–R interval of the extrasystole is prolonged When auricular extrasystoles appear very early in *diastole, they may be blocked on their journey to the ventricle, that* is, they are not followed by a ventricular complex A bradycardia may develop as the result of numerous blocked auricular extrasystoles Owing to the constant variation of tone of the cardiac nerves and the varying excitability of the conduction tissue blocked as well as normally and aberrantly conducted auricular extrasystoles may continually *follow one another and very peculiar variations may thus develop in the electrocardiogram*

In Fig 101 three tracings all Lead II, are reproduced from three different cases with auricular extrasystoles The time record *in the upper is 1/5th of a second, in the two lower, 1/25th of a* second

In the first tracing after the first normal beat, an auricular extrasystole appears with a peaked abnormal P wave, this is followed by an entirely aberrantly conducted ventricular complex After a post extrasystolic pause a premature abnormal P wave again appears, perhaps through an alteration of the vago sympathetic tone, this is not conducted to the ventricle, that is, it is blocked At the end of the tracing a third auricular extrasystole may be seen, *it is conducted slowly to the ventricle (pro*

longation of P–R interval) and also spreads abnormally within the ventricle (abnormal ventricular complex)

In the second tracing an auricular extrasystole follows each normal beat (auricular bigeminy) The first auricular extrasystole is blocked, the following three are conducted abnormally in varying degrees

In the third tracing two auricular extrasystoles appear after three normal beats The P wave of the first is seen distinctly as a notch in the T wave of the third normal beat This extrasystole is so premature that the conduction system has still not completely

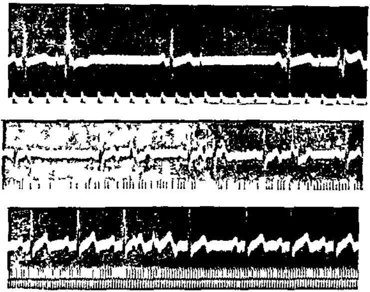

Fig 101 Examples of aberrantly conducted and blocked auricular extrasystoles

recovered and the following ventricular complex is conducted slowly and entirely abnormally within the ventricle Its initial deflection is very small Immediately after the T wave of the first auricular extrasystole one sees the inverted P wave of the second auricular extrasystole This spreads in the ventricle in a normal manner

In Fig 102 two normal beats may be seen, and then a series of five auricular extrasystoles with inverted P waves of which the first four are conducted normally, and the fifth is blocked After the following normal beat another blocked auricular extrasystole occurs

After the inverted P-waves of the blocked auricular extrasystole one sees a flat sluggish positive wave in the electrocardiogram, this has been mentioned earlier as the terminal deflection of the P wave (the "Ta"-wave)

It is evident that the variations in the electrocardiographic picture of auricular extrasystoles which have been mentioned are actually the result of their premature appearance since in the same

Fig 102 Multiple auricular extrasystoles some of which are blocked

case one can often observe that the earlier they appear in diastole the more poorly are they conducted to and within the ventricle

Interpolated auricular extrasystoles are not observed since auricular extrasystoles disturb stimulus formation in the sinus node *Isolated observations of cases reported as such are open to serious* criticism

Mitral stenosis was present in the patient from whom Fig 103 was recorded This explains the large, broad and splintered P waves

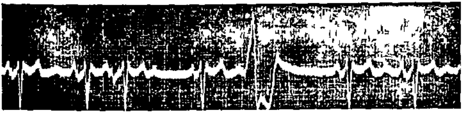

Fig 103 An auricular and a ventricular extrasystole as well as distinct *U waves in Lead I*

and the deep S waves in Lead I After the T wave a distinct U-wave is found, which should not be confused with a P wave Moreover, one sees a disturbance of rhythm which is produced by an auricular *and* a ventricular extrasystole

Auricular extrasystoles with continually changing P waves are a sign of organic myocardial disease just like ventricular extra systoles originating from several foci Digitalis never produces auricular extrasystoles

Mode of Origin of Extrasystoles

Not much is known concerning the finer mechanism of the development of extrasystoles For years the formation of the extra systolic stimulus has been considered different from slow automatic stimulus formation A distinction was made between the homotypic (Hering) or homogenetic (Lewis) and the heterotypic or heterogenetic development of the stimulus The first is intended to be comparable to the physiological formation of a stimulus in the sinus node, while the second is said to develop as the result of some abnormal type of stimulus production To be sure, this distinction has in recent years been abandoned by most authors (Lewis, Rothberger Wenckebach) and the automatic and the extrasystolic stimulus formation were considered as essentially similar events and the various extrasystolic allorhythmias were ascribed only to interference of the existing basic rhythms with an extra stimulus rhythm

Recent investigations however, have shown that not everything which impresses one clinically and electrocardiographically as an extrasystole develops in the same way, and that an explanation which may be valid for a special case does not apply to all extrasystoles The usual definition of an extrasystole a premature contraction which appears in the midst of another rhythm, has too many exceptions

A vast majority of extrasystoles have *one* common property which must be considered in every attempt at explanation the firm time relation between the extrasystole and the preceding beat, which remains constant despite the greatest variations in the basic rhythm Proof has now been obtained that the firmly bound extrasystole *is released only through a foreign stimulus* and does not develop independently

In older discussions at times one finds explanations of the origin of extrasystoles which now seem quite fantastic Thus, one author observed them especially in association with mitral stenosis and explained them in the following manner since the blood cannot be ejected from the dilated left auricle by *one* systole owing to the stenosis, a second, the extrasystole, follows soon after the first Another author noted that extrasystoles were especially common in heart block, according to him the extrasystole has the task of preventing too marked a bradycardia and represents a protective mechanism which the heart possesses

In addition to the explanations of extrasystoles by homo- or heterogenetic stimulus formation, numerous other investigators, especially de Boer, have attributed the extrasystole to a circus movement of the excitation wave If the normal stimulus has spread

over the heart at times it is able to travel once again over the auricle or ventricle without becoming arrested A large number of possibilities for the occurrence of this circus movement have been discussed

When a mammalian heart has been cautiously treated with aconitine it can be shown that a very regular bigeminy occurs at a definite stage of the intoxication If during this state artificial extrasystoles are produced in various ways and are induced from the right or left ventricle then *the same kind* of extrasystole follows them as develops spontaneously after every normal beat In Fig 104 such an experiment is reproduced After every normal beat two left ventricular extrasystoles follow (Lead III) and two right ventricular extrasystoles produced by electrical stimuli are also visible They are followed by two left ventricular extrasystoles that are similar to those following the normal beats

Irrespective of whence the releasing beat emerges *it produces the same type of extrasystole* In other words the extrasystole does not develop independently but is precipitated by the preceding beat

Since the extrasystole always has the same form regardless of the location of the releasing beat it may no longer be assumed that the extrasystole originates by a wave of excitation failing to come to rest and spreading again over the heart If this explanation were valid then the second circle would be obliged to travel in a different direction and the form of the extrasystole would change when the spread of the excitation wave of the releasing beat altered

Another observation tends to support these objections In aconitine poisoning the interval between the extrasystole and the preceding beat which precipitates it continually remains the same as long as an undisturbed regular bigeminy or trigeminy persists If for instance experimentally produced left ventricular bigeminy was present and a left sided beat was artificially produced by stimulation in the midst of diastole then the coupling of the extrasystole to this left-sided beat became shorter This is comprehensible since a stimulus developing in the left ventricle more rapidly activates an extrasystolic centre lying in the same ventricle and precipitates the extrasystole earlier However if a right sided beat was artificially produced it was also followed by the same type of left extrasystole Since the stimulus developing in the right ventricle requires a longer time to reach the extrasystolic centre in the left ventricle in these circumstances the coupling was longer The increase in time (0 04–0 05 second) between the releasing beat ' and the following extra systole corresponds to the interval necessary for the stimulus to pass

FIG. 104. Trigeminal rhythm after the administration of aconitine to a dog. Two right ventricular extrasystoles (fourth and tenth beats) are followed by two similar left ventricular extrasystoles as the normal beats.

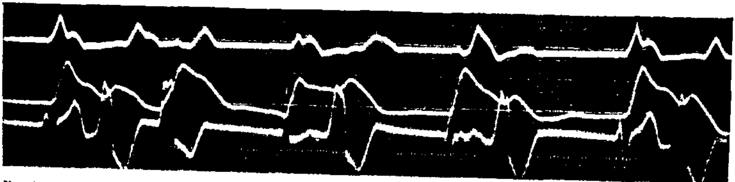

FIG. 105. Right ventricular extrasystoles after aconitine. The extrasystoles follow after normal beats or left ventricular beats. The interval between the releasing beat and the extrasystole is 0.01–0.05 seconds longer if the extrasystoles appear after a left ventricular beat

from one ventricle to the other (see discussion of bundle branch, block, p 49)

In Fig 105 the coupling of the extrasystole which followed a right sided beat correspondingly was 0 04–0 05 second longer than that of the other extrasystoles The coupling is longer in proportion to the distance which the wave of excitation of the releasing beat must travel until it reaches the extra stimulus centre

This observation not only opposes the assumption of the circus movement (re-entrant beat) theory as an explanation of the extra systoles, but also constitutes an objection to the conception that extrasystoles are released independently and automatically The same results could be obtained by means of another experimental arrangement In an experiment a left sided extrasystole was present as shown in Fig 106, the coupling continuously had the same length If, by pressure, the right bundle branch was rendered temporarily incapable of conduction, then the coupling remained unaltered despite a decided change in the form of the normal beat (upper right tracing) After the right bundle branch had recovered the left bundle branch was severed completely (lower left tracing) The form of the left extrasystole remained unchanged, but the coupling increased because the releasing wave of excitation was compelled to travel a longer path over the right ventricle before it reached the extrasystolic centre, the coupling was 0 04–0 05 second longer If at this time the right bundle branch was also completely severed a complete block existed Automatic ventricular beats occurred originating as they commonly do in experiments of this kind from the right *and* left ventricle The left extrasystole remained unchanged, only the coupling was correspondingly longer when it followed a right sided beat

In other words, the length of the coupling is dependent upon the moment when the releasing stimulus reaches the extrasystolic centre

Moreover, further investigations indicate that the extrasystole actually originates in a sharply circumscribed area of the ventricle When in these experiments a bigeminy was produced and the extrasystoles originated in a definite known part of the ventricle warming of this point increased the number of extrasystoles and even caused a paroxysmal tachycardia without any change in the form of the extrasystoles

On the basis of our present knowledge the development of extra systoles may be formulated in the following manner if a wave of excitation originates in some optional area and spreads to an abnormal centre, this abnormal centre may at times respond by

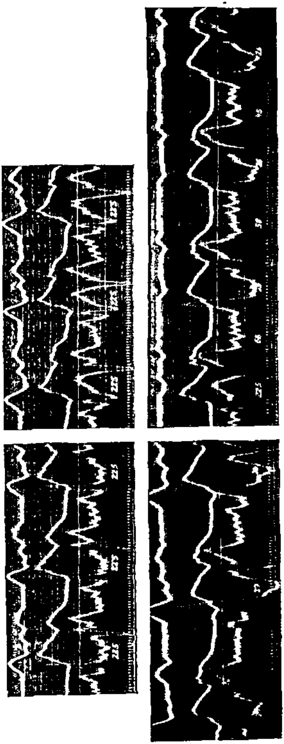

FIG. 106. See description in text.

several discharges and not necessarily by only one, just as a nerve and muscle preparation sometimes responds to a single stimulus by many discharges or contractions According to the circumstances of the case, this abnormal formation of stimuli may appear rapidly or slowly, at rare intervals or often

Clinical Aspects and Treatment of Extrasystoles

Every extrasystole produces a considerable disturbance of cardiodynamics Extrasystoles are premature and appear early in diastole. Often they are so premature and the pause preceding them is so short that an effective filling of the ventricle is impossible The more prematurely the extrasystole occurs the smaller the stroke volume For this reason it is difficult to feel the pulse wave of the extrasystolic contraction, if the extrasystole is very premature, no pulse may be palpable in the peripheral arteries (frustrane contraction) If a bigeminy is present the physician who palpates the pulse may think that he has discovered a bradycardia ('pseudo bradycardia') In other words during the extra contraction less blood enters the arterial system and there is stasis behind the heart in the venous system But since the post extrasystolic interval is longer than a normal cardiac cycle of the same patient, the stroke volume of the first normal beat following the extrasystole is much larger, the related pulse wave much stronger If the extra systole advanced too little blood into the arterial system, the next beat compensates for it, thus stasis behind the heart, in the auricles and great veins is avoided and sufficient blood is propelled toward the organs Since a complete balance occurs it becomes comprehensible why patients who have suffered continuously from extrasystoles for many years show no circulatory disturbances While the single extrasystolic cardiac contraction may advance less blood the minute volume on which all depends remains normal But it will be understood that disturbances of the circulation develop when many extrasystoles occur without post-extrasystolic pauses (multiple extrasystoles many interpolated extrasystoles)

Fig 107 reproduces a pulse curve above and an electrocardiogram in Lead III below An auricular extrasystole appears after two normal beats Although it does not occur very prematurely, its pulse wave is much smaller than that of the normal beat If a ventricular extrasystole had occurred at the same time in diastole its pulse wave would have been still smaller, in auricular extra systoles the auricular contraction preceding the ventricular systole

improves ventricular filling The first normal beat appearing after the extrasystole has a much higher pulse wave than all other normal beats, since a longer diastole precedes it

Since the patient had a poor myocardium a brief pulsus alternans appeared after the extrasystole This is a very common phenomenon after extrasystoles In Fig 107 the pulse which follows the high pulse wave is small the next higher, etc In the electrocardiogram one sees only a slight alternation of the form of the T wave Very often nothing abnormal is seen in the electrocardiogram of patients presenting *pulsus* alternans since the phenomenon develops from a disturbance of con tractility and this does not influence or only slightly influences the form of waves of the electrocardiogram (electrical alternans, see p 17)

The *clinical diagnosis* of extrasystoles is easy by auscultation without the employment of graphic methods In an otherwise

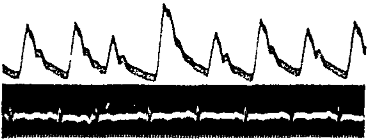

Fig 107 Above pulse tracing and below, the electrocardiogram of an auricular extrasystole electrical alternans in the electrocardiogram after the extrasystole pulsus alternans in the pulse tracing

regularly contracting heart a premature contraction followed by a long pause is heard If bigeminy exists, that is, continuous "twin contractions," usually with some attention the diagnosis is possible even when other disturbances of rhythm are present (for example, auricular fibrillation) A clinical differentiation between auricular and ventricular extrasystoles cannot be made

Multiple extrasystoles appearing at irregular intervals are at *times mistaken for auricular fibrillation A differentiation is possible* by means of an exercise test or the inhalation of amyl nitrite Extra systoles vanish during and immediately after effort and during the effect of amyl nitrite while the arrhythmia of fibrillation is augmented by work

The earlier the extrasystole appears, the more inadequate the filling of the ventricle and the louder the first sound (sometimes it is "thumping,' as in mitral stenosis) The second heart sound is softer to the same extent, it may even vanish since the ventricular

filling becomes progressively impaired with increasing prematurity of the extrasystole, and if extrasystoles occur very early the aortic valves are not even opened during systole In cases of this sort after the two normal sounds of the normal beat preceding the extrasystole, only the first loud sound of the premature contraction is heard In a bigeminy three sounds continuously are audible so that confusion with gallop rhythm may occur

Not rarely extrasystoles are absent at the time when the patient is examined or when the electrocardiogram is recorded Excitement, effort, everything which shortens diastole or produces a tachycardia may cause the extrasystoles to vanish If one desires to make certain the diagnosis, the extrasystoles may be elicited by three methods (1) extrasystoles appear after exertion, for example, after bending the knees several times, (2) they appear after the inhalation of amyl nitrite, (3) they appear during or after pressure on the carotid sinus During and immediately after work, immediately after the inhalation of amyl nitrite (a few drops inhaled from a cloth as in angina pectoris), the extrasystoles are often absent when they previously were present When some time elapses after these procedures, when the heart has again become quiet and the depressing vagus again has the upper hand, they frequently reappear With the carotid sinus pressure test, extrasystoles occur during the exertion of the pressure

Usually the extrasystoles produce no unpleasant sensations, repeatedly one observes for months or years extrasystoles, even bigeminy, which have not affected the patient or given rise to any symptoms In many patients attention is for the first time drawn to a disturbance of cardiac rhythm quite accidentally, e g, by feeling their pulse, by observation of the cardiac rhythm in the stillness of the night (pulse in the ear which rests on the pillow), or, unfortunately, by the examining physician

At times, however, extrasystoles produce considerable distress right from the beginning In rare instances the extrasystole itself is felt as an unpleasant thrust The patient gives a very characteristic description of his complaints, so that the diagnosis can be made solely upon the basis of his report, he mentions the sensation of a sudden thrust in the chest as though something moved inside, indeed, according to the imagination, education and occupation, various descriptions are advanced, but they all imply a short rapidly running thrust like movement in the region of the heart Women who have borne children may speak of a sensation which recalls the movements of the fœtus during pregnancy,

the hunter of a rabbit dodging the pursuing hound, the athlete of a heart which jumps, etc A desire to cough with every extrasystole is a not uncommon result of an interesting reflex This cough comes "by itself" without irritation

More frequently the *pauses* after the extrasystoles are felt unpleasantly The patient has the sensation as if *the heart stood still* He anxiously awaits to see whether or not it will beat again It is not quite understandable why the relatively brief post-extrasystolic pause produces this sensation since disproportionately longer periods of cardiac standstill (for example in disturbances of conduction) are not perceived by the patient It is very probable that still other factors additional to the pause are responsible for the sensation of cardiac standstill

Among the most common sensations is the unpleasant thrust felt by the patient during the normal systole which comes after the extrasystole It produces similar sensations and is described with the same vivid terms as the symptoms produced by the extrasystole itself This fact is comprehensible in view of the larger stroke volume which the post extrasystolic contraction propels Corresponding to the longer diastole the filling of the ventricle is increased and the contraction is very powerful In cases of insufficiency of the aortic valves or in cases of hypertension in which the left ventricle is enlarged and even with regular cardiac activity a large stroke volume is advanced the contractions after extrasystoles are particularly unpleasant

Extrasystoles occur at all ages Repeatedly they have been heard during auscultation of the fœtal heart sounds Electrocardiograms taken immediately after birth have confirmed the diagnosis (Hecht Antoine) Usually auricular extrasystoles are found in children during the first years of life

Extrasystoles are not always a sign of an organic heart disease Very often they occur in healthy individuals In some cases it is possible to determine the cause of the extrasystoles For example, some poisons precipitate extrasystoles In animal experimentation a great many come under consideration but in practice three are mainly responsible (1) digitalis whose significance has already been mentioned , (2) epinephrine and related substances (ephedrine etc), for example they produce extrasystoles not rarely in cases of asthma, through the stimulating action of these substances upon the sympathetic nervous system and on the centres in the heart muscle , (3) likewise nicotine, that is, smoking may produce extrasystoles This form of extrasystole is not common, and

its incidence varies in different countries, according to the strength of the tobacco consumed Discontinuance of smoking leads to the rapid disappearance of these extrasystoles

In other cases the extrasystoles appear, as the patient reports only upon excitement only some time *after* exertion, only in the right or left lateral position, only during deep inspiration or expiration, only with severe constipation after abundant meals, in cholelithiasis and in cases of cholecystitis or before the menstrual period Their origin by way of reflex is better appreciated since in animal experiments, under certain conditions, workers have succeeded in producing extrasystoles by stimulation of the carotid sinus (Schott) and since extrasystoles can also be produced by an " extrasystolic centre in the central nervous system

There is never one single " cause for the appearance of extrasystoles , rather a constellation of various factors is necessary In the vast majority of cases the extrasystoles appear and vanish without their cause having been ascertained

Extrasystoles develop through an *abnormal* process of stimulus formation But it is certain that no one has the right, simply on the basis of the fact that extrasystoles are present, to assume the existence of a disease of the heart The number of specific fibres in the auricle and in the ventricle is very large How easy then is it for one of these fibres to show a slight anomaly an abnormally increased or decreased permeability of the cell membrane for certain electrolytes an increased irritability or an unusual manner of reaction to an external stimulus ! But a heart is not necessarily sick in which *one* cell or cell group is abnormal Also in other large groups of cell aggregates in the organism one or another abnormal cell may well be found without the organ s to which this cell belongs being designated ' diseased " One must concede that nowhere else is such an anomaly so easily recognized as in the heart here a stimulus formed by an abnormal centre, if it is above threshold excites the neighbouring fibres then the entire muscle and thus leads to a distinct and visible disturbance

If extrasystoles appear suddenly in a patient, we can at first merely conclude that a disturbance exists in a sharply defined area

After what has been said, it may consist simply of an anomaly of *one* cell or group of cells and the extrasystole is without significance Within certain limits, it is then immaterial for the appraisal of the case whether rare or numerous extrasystoles are found

But in addition of the one cell whose abnormal activity one

observes in the form of an extrasystole, many other cells, for example those of the common muscle may be abnormal, so that one must speak of a disease For this reason, every patient in whom extra systoles appear must be subjected to a careful examination and if the result of the investigation is negative, the individual should be observed for a time It is particularly important to proceed in this manner when extrasystoles appear in diseases which experience shows damage the heart muscle regularly or frequently, such as diphtheria, pneumonia, coronary sclerosis or thrombosis, and many other conditions In many cases only after long observation can one decide whether or not a purely local disturbance exists which may be safely disregarded or whether the extrasystoles indicate that a cardiac disease has developed which otherwise would not have been detected

If ventricular extrasystoles have ventricular complexes of various forms an organic heart disease is present

If a patient consults a physician with the report that the complaints have existed *for years*, then, even with the negative result of a single thorough examination, it may be assumed that a harmless disturbance exists in a healthy individual

After the preceding remarks a discussion of the *prognosis* of extrasystoles may be omitted We noted that extrasystoles often are merely harmless nonsense of the heart' (Wenckebach), but sometimes they may be the first sign of a serious disease The judgment of the individual case depends upon what can be shown to be the precipitating cause of the extrasystole

The *therapy* of extrasystoles varies according to the status of health of the patient and the cause of the extrasystoles If the patient is not aware of his extrasystoles and the examination and observation demonstrate a normal heart, it constitutes a serious error to direct the attention of the patient to his cardiac irregularity The layman is inclined to regard the occurrence of any irregularity in the constant regular rhythm of the pulse as a sign of serious disease This kind of extrasystoles should not be treated and should be disregarded altogether

If a patient has *felt* his extrasystoles for only a short time it is again important to ascertain (careful examination, observation) whether it is a case of extrasystoles in a healthy person or in a person suffering from heart disease If all the findings are normal, if there is no reason for assuming the presence of heart disease, if in the electrocardiogram the extrasystoles do not vary in shape, then the harmlessness of the disturbance should be explained to the

patient and an endeavour made to convince him that he can lead an entirely normal life The physician is supported in his argument by the fact that while physical exertion is always unpleasantly felt by the organic cardiac patient, it is not only possible but even causes *the extrasystoles to vanish in the patient with a healthy heart*

If the physician omits this explanation, if he lacks the necessary knowledge and emphasis, if he speaks—as happens not rarely—of a "slight heart disease," and advises care and avoidance of exertion, the patient merely sees his own conception strongly confirmed, he observes himself with increasing anxiety and soon develops a severe neurosis which can be influenced only with difficulty Thus a tormenting state may develop from a harmless phenomenon But if one succeeds in convincing the patient that his extrasystoles are without significance, he soon learns to disregard both them and the *symptoms to which they give rise*

The psychotherapeutic measures of the physician should not merely consist in "talking the patient out of his disease," but rather in exercising his entire medical authority to induce the *patient as a healthy individual to lead his life accordingly Nothing will convince a patient so completely and so quickly that the* cardiac irregularity is harmless than the absence of any prohibitions or restrictions The patient must be told not to count the pulse, not to "await the thrust," not to count how often it occurs in a minute

In other words, the harmless *extrasystoles of the healthy individual* require no medical treatment For the most part the result of therapy depends upon the success achieved in having a favourable influence upon the patient and *convincing* the patient, the extrasystoles are not treated, but simply an attempt is made to insist *that the patient need not trouble himself about them, this is not* always easy, especially in neurotic individuals

If extrasystoles appear in connection with an organic cardiac disease, the type of therapy will naturally be influenced by the associated disease As far as possible, *one will treat a coronary* sclerosis, and in diphtheria or coronary thrombosis will keep the patient quiet, in a digitalis bigeminy the procedure is adapted according to the rules advocated earlier (p 179) If definite precipitating factors or causes are found an attempt *should be made to remove them Often the removal of a severe meteorism*, of a high position of the diaphragm, of a severe constipation, permanently abolishes the extrasystoles

Medicinal therapy can be omitted in the majority of cases for the additional reason *that it aids only during the period of its use*,

when the treatment is discontinued the extrasystoles reappear and the patient becomes more disquieted than ever Obviously drugs cannot indefinitely be administered

Drug therapy is necessary when the extrasystoles multiply in a threatening manner or when they cause a great deal of distress so that it seems advisable to abolish them at least temporarily and thus convince the patient that we possess agents which can help him But it is always advisable to inform the patient prior to the treatment that the extrasystoles will reappear after the cessation of treatment He may carry the capsules with him and may take them when the extrasystoles are very distressing

Since in cases of mitral stenosis auricular extrasystoles frequently precede auricular fibrillation it is advisable when extrasystoles appear to administer quinidine prophylactically in these and other patients who tend to fibrillate The same rule holds if ventricular extrasystoles develop in a case of coronary thrombosis they must be treated with adequate doses of quinidine since otherwise they may multiply and produce a ventricular tachycardia occasionally ventricular fibrillation may develop The fear of giving quinidine by mouth to patients with damaged hearts is not founded in fact so long as the doses employed do not exceed those mentioned below

Quinine may be mentioned as the most effective remedy for extrasystoles It was recommended by Wenckebach (1914) as a substance which reduces the excitability of the heart Through this property and the prolongation of the refractory phase inhibition of stimulus formation extrasystoles are almost always removed if adequate doses are administered

In the treatment of extrasystoles as in all cardiac therapy one should not proceed purely schematically with the same dose in every patient Rather the amount of the drug should be adapted to the requirements of the individual case this is readily accomplished since the result of therapy can be very easily controlled by auscultation or by palpation of the pulse Capsules or pills of 0 1 gm (1½ grs) of quinine hydrochloride (or the more effective quinidine sulphate) are prescribed four to five capsules are administered It should be observed that the first dose is taken as early as possible in the morning and the last late at night If these amounts which are certainly very small do not suffice then 0 1 gm (1½ grs) more is taken daily until the smallest amount is determined that is sufficient to prevent the appearance of extrasystoles If one finds that the extrasystoles still occur with 7 × 0 1 gm of quinidine distributed

throughout the day and none occur when 8 × 0 1 gm are administered then eight capsules are given and after some time an attempt is made to reduce the amount at times success is then attained with much smaller doses

A second drug which can remove extrasystoles is digitalis At first it may seem contradictory that an agent which very frequently produces extrasystoles can be utilized for their removal But as emphasized earlier (p 177) digitalis extrasystoles develop only under definite conditions They are easily differentiated in the electrocardiogram from the harmless extrasystoles of the healthy individual They alone contraindicate digitalis therapy However extrasystoles which are not the result of digitalis, as a rule vanish upon the administration of this remedy Very often small doses of digitalis suffice to suppress the extrasystoles only in some cases with multiple extrasystoles are larger doses necessary One disadvantage of digitalis therapy often consists in the fact that the patient realizes that digitalis is a remedy for cardiac weakness and is alarmed by the use of the drug For this reason it is suggested that one prescribe only those preparations of digitalis whose name is meaningless to the patient The digitalis must be administered especially when because of hypersensitivity or allergy quinidine is poorly tolerated One may employ active purified preparations of squill in place of digitalis

The combination of small amounts of quinidine with digitalis is very effective and widely used for example in the form of Wenckebach s pills these also contain some strychnine as a tonic —

Quinidine sulphate	4 0 gm
Pulv fol digitalis (assayed)	2 0
Strychnine nitr	0 06
Mass pil q s pil No C	
S Three to six pills daily	

According to the circumstances of the case this composition may be altered With persistent extrasystoles it is advisable to increase the dose of quinidine but at times even when no cardiac failure is present an increase in the amount of digitalis is advantageous

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FIBRILLATION AND FLUTTER

Introduction

If by strong induction shocks the auricle of a dog heart is stimulated 100–200 times a minute, according to the rate of stimulation it will contract 100–200 times a minute and in the electrocardiogram a corresponding number of P-waves will appear; also with a rate of stimulation of about 300 per minute every stimulus will yield a response But if the rate is further increased, one or another wave of excitation will spread abnormally in the auricles since some of the muscle fibres already become fatigued and no longer can conduct all stimuli, the P-waves will no longer display the same form and some appear abnormal If the rate of stimulation is increased to rates above 600, no single muscle bundle of the auricle will respond to every stimulus, the spread of excitation in the auricles will become entirely irregular, and the P-waves exhibit a continuous variation of form *With an increase of rate beyond a certain limit the auricular electrocardiogram becomes increasingly irregular.*

Through events which will be mentioned later, very high auricular rates may appear in man The condition in which the auricles are stimulated very rapidly, approximately 300 times a minute, and in which in the electrocardiogram the auricular waves are very frequent but of regular form, is called *auricular flutter.* If with higher rates the auricular waves become irregular, *auricular fibrillation* is present The transitional form between the two conditions is known as *impure flutter* which may be assumed to be present when the auricular electrocardiogram shows only very slight irregularity which is not sufficiently marked to be called fibrillation. The auricular waves associated with these high rates are not called P-waves but "F" waves (fibrillation-flutter waves).

If in the electrocardiogram one sees *regular F-waves* in place of the single P-waves which normally precede every ventricular complex, the diagnosis is auricular flutter; if irregular F-waves are found, auricular fibrillation is present. Impure flutter lies between these two states It is not the auricular rate, but the *form* of the auricular waves which is significant for diagnosis With a very good state of the auricular muscle, one will find regular F-waves, that is, flutter sometimes even with rates of 350–400. With damaged,

functionally impaired muscle fibres the F waves may be irregular even with rates of stimulation of 300 or less and then auricular fibrillation is present

Although auricular fibrillation had been known for a long time in animal experiments it was discovered in man in 1909 by Rothberger and Winterberg as well as by Lewis

Auricular flutter in man was described for the first time by Jolly and Ritchie in 1910

The Electrocardiogram in Auricular Flutter

Just as the appearance of P waves in sinus rhythm so the appearance of flutter waves varies from case to case The rate of the fluttering auricles often approximates 300 per minute but it may be as low as 200 and in rare cases may rise to 400 (Under the

a

b

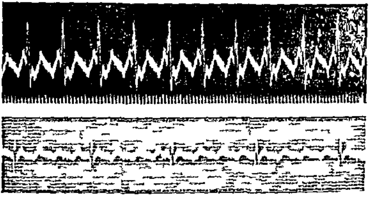

Fig 108 Two examples of auricular flutter above a 2 1 block below a 4 1 block

influence of therapeutic measures flutter rates below 200 may be observed) With higher auricular rates one F wave adjoins another so that the electrocardiogram shows an unbroken line of waves a continuous rise and fall like the teeth of a saw With slower flutter the individual F waves are separated by small isoelectric intervals As will be considered later the differentiation between auricular flutter and auricular tachycardia is then very difficult The F waves may be large or small pointed or round positive or negative Often they are invisible in one or more leads usually in Lead I

Fig 108a shows auricular flutter with 2 1 block (Lead II) The form of the F waves is remarkably regular and they appear at the rate of 300 every second F wave is bisected by a QRS complex Fig 108b shows auricular flutter with a 4 1 block

Fig 109 reproduces a three lead electrocardiogram of a case of auricular flutter An unbroken chain of regularly formed equal F waves are evident and may best be seen in Leads II and III The F waves are scarcely visible in Lead I and give the impression that P and T waves are present, on superficial inspection Lead III also creates the same impression On closer examination, however one finds that the P–R interval is very short, that the T wave occurs too early after the QRS complex and that the P and T are identical in shape Moreover, measurement shows that both waves are equidistant from each other There is a 2 : 1 block, save for a 3 : 1 block at the end of Lead I

Fig 109 Auricular flutter in the three leads (2 : 1 block)

The rate and rhythm of the ventricles has no significance in the diagnosis

In auricular flutter the ventricles may beat in every conceivable ratio to the auricles and with any possible rhythm Occasionally every auricular beat is conducted to the ventricle so that not only 300 auricular contractions but just as many ventricular beats occur Fortunately such a high rate of the ventricle is uncommon since it presupposes a very good state of the conduction system and a low vagal tone Very often only every second or third auricular stimulus is conducted to the ventricles Then a 2 : 1 block (half rhythm) exists or a 3 : 1 rhythm the ventricular rate being one third of that of the auricles But since 4 : 5 : 6 : 1 block may occur, a very marked bradycardia may be present in flutter The degree of block is not always constant Very often it changes so that the ventricles

contract irregularly, by virtue of the fact that this irregularity of conduction often recurs every possible allorhythmia may develop and upon clinical examination erroneous diagnoses occur all too easily

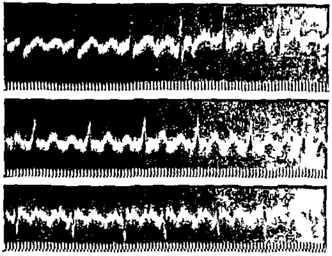

FIG 110 Auricular flutter with 2 1 and 3 1 block

Fig 110 was obtained from a case of auricular flutter and presents varying 2 1 and 3 1 block The flutter waves are very

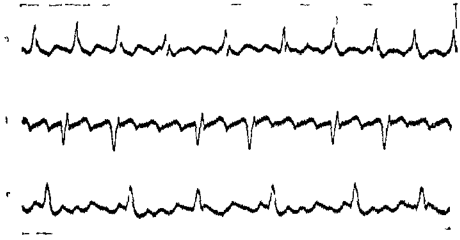

FIG 111 Examples of auricular flutter with variable block

indistinct in Lead I The diagnosis would not be easy from Lead II alone since sinus bradycardia with normal P and T waves might be assumed Measurement with dividers, however awakens suspicion

that regular flutter exists although only *one* F wave that in form suggests a P wave is visible, while the other flutter waves are hidden in the QRS complex and the T wave

In Fig 111a the auricular flutter with 2 1 block similar to Fig 109 is again shown in Lead II, since the F waves are concealed in the initial and terminal deflections, the diagnosis is not easy Slowing of the ventricle as the result of carotid pressure (in the middle of the tracing) permits the regular F waves to appear Fig 111b shows flutter with varying 4 1 and 2 1 block so that the diagnosis of bigeminy might be made upon auscultation (note also the deep Q waves in Lead III) In the other tracing of Fig 111c there is

Fig 112 Impure auricular flutter

auricular flutter with 3 1 and 2 1 block Since at some places the T waves coincide with F waves, a remarkable tracing develops

Fig 112 shows another instance of auricular flutter But since the F waves show slight variations in form, it may be called "*impure flutter* The appraisal of the appearance of the F waves is possible only where they appear in the middle of diastole and are not deformed by association with a QRS complex or a T wave The low voltage in Lead I does not necessarily indicate only a low position of the diaphragm with a "dropped heart" since it is also found in the early phases of a developing dextrocardiogram the R wave has already become low, but the deep S wave has not as yet appeared QRS-complexes of this type are common in mild mitral stenosis

Difficulties are encountered in diagnosis when the F waves as in Figs 109 and 110 are hidden in the QRS complexes and T waves

Thus there are cases of auricular flutter with 3 1 *block in which* one F-wave falls within the QRS complex, a second is hidden in the T-wave and the third occurs in ventricular diastole and looks like a *normal* P-wave *so that ordinary sinus rhythm is assumed* and the flutter is overlooked (Fig 110, Lead II)

Likewise, when flutter with 2 1 block is present one may at times mistakenly think of a sinus tachycardia since regular, fast P–QRS–T complexes are seen (Fig 113) But after some practice it becomes easily apparent that the P–R intervals are remarkably short, the T waves lie very near to the QRS complexes, the P and T waves are identical in form and appear equidistant from each other A pair of dividers must always be employed to determine whether or not the distance between two presumed F waves are equal *Measurement will often immediately reveal that no flutter exists and in other* cases confirms the *possibility of flutter* In this connection, however, it should not be forgotten that also in harmless tachycardias, if they attain a certain rate, the P–T interval may be just as long as the T–P interval Reference will be made later to a method of differentiation between the various forms of tachycardia and flutter (p 255) Here it need merely be said that the electrocardiographic diagnosis is not always possible

Fig 113 (*Lead II*) Auricular flutter with 2 1 block

Very often the diagnosis can be made only from *one* lead since the F waves are not distinctly visible in the others A chest lead (to the right of the sternum, p 150) at times is necessary for the diagnosis

It is not always easy to say which F-wave is conducted to the ventricle This holds particularly for cases with variable auriculo ventricular conduction But this question has merely theoretical and no practical interest

The Electrocardiogram in Auricular Fibrillation

The fibrillation waves in the electrocardiogram may be extremely variable in their appearance There may be very large coarse F-waves or very small, scarcely visible, " fine " fibrillation waves For practical purposes it is unimportant whether large or fine fibrillation exists, the difference is purely external The F-waves

are often absent in all leads In these cases the diagnosis is made from the complete ventricular arrhythmia and the absence of P waves If one desires to confirm the diagnosis an attempt to record auricular waves by means of a chest lead is recommended

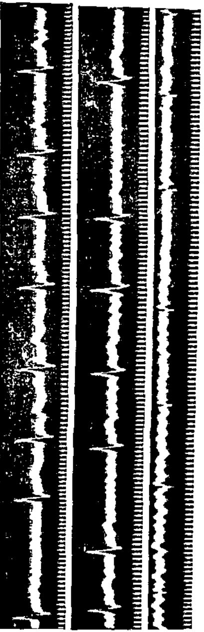

Fig 114 Auricular fibrillation

Fig 114 shows auricular fibrillation There are no P waves in their place one sees continuous F waves which are irregular in form (especially distinct in Leads I and II) The ventricles contract entirely irregularly The ventricular electrocardiogram is in general normal actually the initial deflection is somewhat plump the *very* small initial deflection in Lead III may appear normally (p 28)

Complete arrhythmia of the ventricle the *delirium cordis* of older medicine is the rule in auricular fibrillation Often one hears the name arrhythmia perpetua but it ought to be avoided since the auricular fibrillation and thereby the arrhythmia may vanish in other words it may not be perpetual As a matter of fact it is not always easy to recognize the arrhythmia With very slow cardiac action and likewise when the rate is very fast often prolonged auscultation and the registration of a long tracing is necessary to demonstrate the arrhythmia distinctly In some cases auricular fibrillation is accompanied by regular ventricular activity then complete heart block co exists

Fig 115 was obtained from a patient with a mitral valve lesion and auricular fibrillation A dextrocardiogram is present without *signs of myocardial damage* *The ventricular rate had been greatly* reduced by digitalis The F waves are easily recognized in the long diastoles

Fig 116 represents an example of a very common type of fibrillation with fine F waves The F waves are perceptible at only a few places and are then indistinct At times they *are entirely invisible* The tracing was obtained from a decompensated patient w i t h m i t r a l stenosis Correspondingly a dextrocardiogram is present The S T segments and the T waves in Lead II and especially in Lead III are depressed below the isoelectric line (result of hypertrophy) This type of tracing is frequently encountered in progressive *mitral lesions with fibrillation*

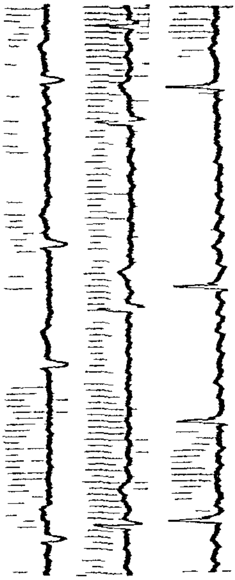

Fig 115 Auricular fibrillation and a dextrocardiogram

In Fig 117 auricular fibrillation with heart block is present The ventricle contracts regularly seventy two times a minute The clinical diagnosis is not possible without the assistance of graphic methods since upon auscultation a regular normal cardiac action was heard Complete heart block is not rare in fibrillation since the conduction to the ventricles of the very rapid and therefore very weak fibrillation stimuli is very easily obstructed if the vagal tone is high or

the conduction system impaired. An *incomplete* block is present in every case of fibrillation.

The arrhythmia of the ventricle in auricular fibrillation is explained by the fact that stimuli from the fibrillating auricle reach

FIG. 116 Auricular fibrillation with "fine" F waves, a dextrocardiogram and oppositely directed terminal deflection

the ventricle very irregularly, in other words, an irregular block exists But the reason for the irregular conduction of the auricular stimuli is at present unexplained.

Since owing to the continuous fibrillation waves the galvanometer string "vibrates," confusion with distorted tracings due to

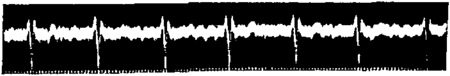

FIG 117 Auricular fibrillation and auriculo-ventricular block.

tremor of the patient, external influences acting upon the stability of the apparatus, etc, is not rare, but is avoidable if care is taken.

Since the ventricular complexes are superimposed upon the fibrillation waves, they can be decidedly altered by the presence of coarse fibrillation This holds for the QRS-complexes which may show very marked differences in amplitude, but especially for the T-waves which may become lost in the F-waves so that their detection is

impossible With high ventricular rates the diastole is very short and there is *no definite T–P interval in which F waves may be* sought In such cases the diagnosis can be made from the changing form of the ventricular *complexes and from the complete arrhythmia*

The difference in the electrocardiographic picture of auricular fibrillation and flutter is evident from Fig 118 where during the registration of the electrocardiogram the auricular *flutter spontaneously passed into fibrillation* In the first part of the tracing there is auricular flutter with 2 1 block, the regular up and down movement of the flutter waves may be seen distinctly and it will be noted that every second *flutter wave is bisected by a ventricular complex* Very suddenly the tracing becomes irregular F waves of irregular shape are observed in the *short diastoles and the ventricular complexes* also become entirely different In fibrillation the ventricular complexes are continuously summated by irregularly shaped F waves and thus the amplitude of the initial and terminal deflections constantly varies

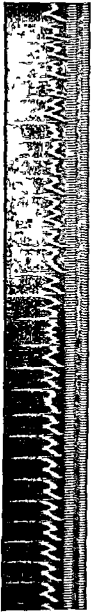

Fig 118 Spontaneous transition of auricular flutter (2 1 block) into auricular fibrillation

Ventricular Flutter and Ventricular Fibrillation

Very fast ventricular tachycardias, the rate *of which is usually about 300 per minute, are* designated as ventricular flutter However it is not rare for paroxysmal ventricular tachycardia with a rate of 250 or less to be described as ventricular flutter Since auricular tachycardia with a rate of about 200 is often designated *as auricular flutter when the auricular waves* show the picture of "the teeth of a saw," this differentiation is logical One must concede that a sharp distinction between paroxysmal tachycardia and flutter often is impossible Some authors regard only those cases as ventricular flutter in which the QRS complexes follow one another in an unbroken chain, no isoelectric line appearing between the individual complexes and these complexes showing no details such as splintering

anatomically demonstrable sinus disease is not the essential factor in the appearance of auricular fibrillation

Auricular fibrillation occurs at all ages of life, but it is much more common in the older age groups (coronary sclerosis) Auricular fibrillation is also found in infants especially after pneumonia, this is comprehensible since it has become known that pneumonia is often accompanied by myocardial lesions

Owing to the complete arrhythmia which even upon prolonged investigation does not reveal any regularity, auricular fibrillation is easy to recognize When the action of the ventricles is slow, at times the irregularity is not immediately evident, moreover, the distinction of fibrillation from multiple auricular extrasystoles of irregular sequence is occasionally difficult In both instances an examination after slight exertion or after the inhalation of a few drops of amyl nitrite assists With an increase of ventricular rate the arrhythmia of auricular fibrillation becomes more distinct, on the other hand extrasystoles will vanish Even without an electrocardiogram an oversight in recognizing auricular fibrillation should no longer occur, the diagnosis can always be made by means of a fairly careful examination

Auricular fibrillation usually develops very suddenly In many cases it appears after increasing numbers of auricular extrasystoles In some cases, therefore, these constitute a warning sign

There are rare cases in which at any time the fibrillation can be precipitated by carotid pressure or by deep breathing

Fig 121 shows at the beginning (left side of the upper tracing) a sinus beat with a depressed S–T segment due to digitalis therapy In the T wave of the first ventricular complex the P wave of a blocked auricular extrasystole is hidden It appeared in the course of a deep inspiration After the second normal complex another auricular extrasystole appeared and introduced a very brief attack of auricular fibrillation A blocked auricular extrasystole also appeared after the fifth and seventh ventricular complexes and two more after the sixth An attack of paroxysmal auricular fibrillation recurred during deep breathing following the auricular extrasystole after the eighth ventricular complex (beginning of the lower tracing) and then became converted into auricular flutter

By deep breathing these attacks could over a long period be reproduced at will Digitalis reduced only the ventricular rate during the fibrillation

In auricular fibrillation the auricles are stimulated 400–600 times a minute With increasing rate the force of the individual contrac

tions becomes progressively weaker and with 400–600 stimuli, such as appear in human auricular fibrillation, contractions are no longer perceptible. If in animal experimentation one looks at the fibrillating auricle from a distance it seems to stand still. Only upon closer examination a slight vibration, a welling and quivering of the auricular wall is seen; this has given rise to the name fibrillation. These extremely fine uncoördinated movements of individual groups of muscle fibres in the auricles naturally are useless for the propulsion of blood. For this reason one can say that in fibrillation the auricles do not function. It is inadvisable to speak of "paralysis"; on the contrary, an activity with an extraordinarily high rate of activation is present.

If the results of auricular standstill are to be understood, one must recall the significance of normal auricular action for the dynamics of circulation. As the result of an auricular contraction a very material amount of blood, about one third of the stroke-volume (20 c.c.), is rapidly ejected into the ventricle. Since shortly before contraction the ventricle rapidly is filled under pressure, the initial tension of its muscle fibres increases and, according to the well known fundamental law of the heart, the contractile power increases correspondingly. If now we take an instance in which the auricles suddenly cease actively

FIG. 121a and b. The lower tracing (b) is a continuation of the upper (a). During [illegible] respiration, there appeared numerous, in part blocked, auricular extrasystoles as well as attacks of auricular flutter and fibrillation. Depression of the S-T segments due to digitalis.

to contract *while the ventricles continue to work at the same rate* the omission of the auricular contraction naturally leads to a decrease of the ventricular filling and to a stasis in the auricles The stasis and increased pressure in the auricles results in an increased amount of blood entering the ventricles during the following diastole so that despite the absence of auricular action ventricular filling is adequate and congestion is avoided

The absence of auricular contraction is without great significance as long as the rate of ventricular contraction is slow and correspondingly long diastoles make possible good ventricular filling When the ventricular rate is high the short diastole does not permit adequate ventricular filling and then auricular action becomes important

From this it follows that patients in whom owing to a high vagal tone or to a poor status of the conduction system not more than 80 stimuli a minute are conducted to the ventricle may feel just as well as normal In such cases the fibrillation is often discovered accidentally and the patient is not aware of it Not rarely during ward rounds in certain cases sinus rhythm is noted on one day and auricular fibrillation on another Many patients feel no worse on the fibrillation days than on others as long as one condition is present *i e* the low ventricular rate If the patient is a neurotic individual who continually feels his pulse the *irregularity* may create apprehension *But when the ventricular rate is low it is devoid of significance* Indeed in discussing extrasystoles it was noted that owing to the variable filling of the ventricle resulting from the irregularity the individual ventricular contractions may have varying force and advance varying amounts of blood but since the short pauses are always followed by long ones each small volume is rapidly balanced by a larger volume and disturbances are thus avoided One may recall that respiratory arrhythmia of the healthy person at times causes greater irregularity than is found in auricular fibrillation with a low ventricular rate Accordingly it may be concluded that *owing to the wonderful adaptability of the heart to varying conditions the arrhythmia alone is not harmful unless it is accompanied by too high or too low a cardiac rate* Only the presence of serious diseases of the heart muscle or of the vessels can disturb this adaptability

In patients with slow fibrillation that is auricular fibrillation with a slow ventricular rate no special treatment is necessary However indulgence in considerable exertion causes these patients to feel worse than individuals with sinus rhythm because the increase in accelerator tone accompanying physical exertion rapidly improves

the conduction to the ventricle and the pulse rate rises Repeatedly, *one encounters patients who have had fibrillation for years and* who, since they have the good fortune to maintain a low ventricular rate, show for years entirely normal circulation without treatment

Unfortunately cases of this type represent the exception As a rule, when auricular fibrillation appears the conduction from the auricles to the ventricles is much better, and the ventricular con *tractions follow one another so rapidly that rates of 180 250* and more, are not unusual The higher the rate the more rapidly severe dis turbances of circulation appear The length of diastole shortens with increasing rates If the auricles were still capable of function they could rapidly press some blood into the ventricles, but since they are ' paralysed the ventricular content always remains reduced With a ventricular rate of about 200 only 50 beats can at times be felt at the periphery Only those ventricular con tractions which develop after longer diastoles produce a pulse most others follow quickly upon one another and are frustrane they advance little or no blood so that the pulse is not palpable This is called a *pulse deficit*, it is proportionately greater the higher the ventricular rate If 50 contractions among 220 produce a palpable pulse the pulse deficit amounts to 170 The size of the pulse deficit is a measure of the degree of circulatory disturbance

Since the diastoles are so short that many ventricular con tractions are ineffective, the blood stagnates in the auricles before reaching the ventricles and less blood is propelled into the arteries But since in a tachycardia the right ventricle ejects too little blood and correspondingly the left receives too little the blood collects only in front of the right ventricle, *i e* in the right auricle and the great veins The neck veins become engorged with blood the patient complains of compression of the neck, the liver swells rapidly, so that pain is felt in the right upper abdomen, and vomiting occurs The patient becomes pale as the filling of the arterial system diminishes and the blood pressure falls *Congestion on the venous side, defective filling of the arterial side of the greater circulation, and absence of stasis in the lesser circulation, are typical of these cases* The higher the cardiac rate the more rapidly this picture develops

All vessels receive less blood than normal, the blood supply of the coronary arteries is also affected so that the heart muscle which owing to the unusually rapid action needs a great deal of oxygen is poorly nourished (p 138) The myocardial injury occasic

(abnormal ventricular complexes) since the conduction system could not sufficiently recover during the short diastoles

In short, there are three different pictures in connection with the appearance of auricular fibrillation If the ventricular rate is approximately 80, the fibrillation produces no change in the sensations of the patient The more the rate exceeds 80 the faster congestive heart failure sets in If the rate is over 100 fainting and even sudden death may result *In other words everything depends upon the ventricular rate* The danger of fibrillation is proportionate to the ventricular rate

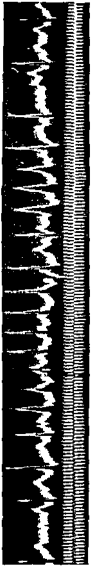

Fig 122 Short attacks of auricular fibrillation with very fast ventricular action and abnormal spread of the excitation within the ventricle

For this reason treatment must be mainly *directed towards the reduction of the ventricular rate* and keeping it low It is well known that excellent success is provided by *digitalis therapy* Digitalis *acts upon the heart* in manifold ways Apart from its action upon contractility it lengthens the so called total refractory phase (the sum of the relative and absolute refractory phases Schellong) and increases vagal tone (probably by way of a reflex over the carotid sinus) Both impair conduction to the ventricles even relatively *small doses of digitalis suffice for these* purposes, these amounts are much smaller than those required in cases of sinus rhythm since the fibrillation stimuli owing to their rate, are also proportionately weaker and much more easily depressed Since this action of digitalis occurs very rapidly and regularly (failure of the slowing effect of digitalis to *appear is very rare and apart from the heart in* hyperthyroidism almost always terminal), the action is spectacular in the decompensated and fibrillating patient Ever since the beginning of digitalis therapy it has been known that digitalis gives its best results in cases with a rapid, irregular and small pulse In such c it the action upon the muscle but the effect upon the re important Digitalis treatment also has the adv *trolled very easily If the rate is between 70–80,*

appear because its presence markedly improves the status of the patient after the use of digitalis

The rule of giving digitalis in auricular fibrillation only as long as the ventricular rate is above 80 holds only when fibrillation without any additional myocardial weakness exists If muscular failure is present this also requires the use of digitalis In rare cases *of auricular fibrillation even in decompensation the* rate is continually low since the A V system is able to transmit only a few stimuli to the ventricle This is advantageous for the patient since a harmful tachycardia does not occur but it also has a disadvantage *in that it leads the physician to withhold the* digitalis therapy that may be necessary for other reasons since the heart is already sufficiently slow without it In other words if signs of decompensation are present digitalis therapy must be instituted even when bradycardia is present

Fig 123 Auricular fibrillation digitalis bigeminy and abnormal automatism

Since in cases of auricular fibrillation myocardial disease often exists and large doses of digitalis must be given very frequently one finds ventricular extrasystoles of varying shape such as have been described earlier (p 176)

Fig 123 shows ventricular bigeminy and trigeminy in auricular fibrillation with a constant change in the appearance of the extrasystoles Since digitalis awakens the automatism of the lower centres abnormal automatic beats (sixth ventricular contraction from the left in the tracing) also appear

In *auricular fibrillation digitalis therapy is* purely symptomatic It is not directed against the fibrillation but against the increased ventricular rate Accordingly the question is raised whether or not one should treat the fibrillation itself This *has become possible since Wenckebach discovered* that quinine abolishes fibrillation frequently one can succeed in defibrillating the patient with quinine and induce sinus rhythm to return Although this therapy yields results in about 70 per cent of the cases it is not recommended for indiscriminate use First of all there are three contraindications which decisively forbid the use of quinine or the more *desirable substitute quinidine*

cumstances the patient's condition can be improved with certainty. Such cases to be sure are not common. A few examples may be cited. A subtotal thyroidectomy has been undertaken in a patient with severe hyperthyroidism and auricular fibrillation but without marked enlargement of the heart. After the operation all the signs of hyperthyroidism vanish but the fibrillation persists and compels continuous digitalization. In such a patient an attempt to defibrillate with quinidine may be made, since thereby a complete restoration of health may be achieved. Another instance: auricular fibrillation appears during the course of pneumonia. The pneumonia subsides but the fibrillation persists. In such circumstances *one has not only the right but the duty to attempt to restore sinus rhythm*. Again, a patient with mild mitral stenosis without marked dilatation of the left auricle begins to fibrillate. In this instance the fibrillation may be harmful because it paralyzes the auricles the contractions of which materially assist in maintaining compensation; indeed auricular activity helps by forcing blood into the ventricle. In these cases the defibrillation is usually not permanent as it is in the two examples cited earlier. However, if it is possible for only six months to maintain full compensation without digitalis this has its advantages.

Prior to quinidine treatment it is advisable to digitalize the patient until there is a ventricular rate of 70–80. Then digitalis may be discontinued and quinidine started. During quinidine therapy strict rest in bed must be maintained since the patient requires supervision the treatment should on principle be carried out only in a hospital.

At first a single dose of 0 25 gm (3¾ grs.) of quinidine is given by mouth, the more powerfully acting quinidine is superior to quinine. *If by the next morning no manifestations of hypersensitivity have* appeared, if vertigo and tinnitus, diarrhœa and erythema are absent, then the actual treatment is started. Since the manifestations of hypersensitivity are not rare and since occasionally they may attain an alarming degree *the administration of a test dose is unconditionally advised*.

The procedure employed is as follows

1st day	3 × 0 25 gm	(3¾ grs.) quinidine
2nd day	4 × 0 25 gm	
3rd day	5 × 0 25 gm	
4th day	6 × 0 25 gm	(or 3 × 0 50 gm.)
5th day	3 × 0·50 gm	
6th day	3 × 0 50 gm	
7th day	3 × 0 50 gm	

Quinidine should not be employed (1) when fibrillation has existed for months or years. The auricles, especially the auricular appendages, stand still. As a rule thrombi are deposited in them and may become detached when sinus rhythm returns and thus powerful auricular contractions are re instituted; severe, not rarely fatal embolism may result. Embolism unfortunately is not a rare event with quinidine therapy.

Quinidine therapy is also omitted (2) if the left auricle is greatly enlarged, even when auricular fibrillation has been present only for a very short time (only a few days). Instances of this kind usually occur in hearts with mitral stenosis so that the danger of thrombosis is especially great and therefore requires special consideration.

Since quinidine is a cardiac depressant and moreover, since very large doses must be given, this therapy cannot be employed (3) when signs of cardiac failure exist or serious myocardial disease must be assumed.

Moreover, even when the contraindications just cited are not present, quinidine need not always be given. As a matter of fact statistics show that while in a majority of cases sinus rhythm is restored by quinidine, the figures also indicate that only transient results are secured. Unfortunately they are often only quite brief in duration. Many cases again begin to fibrillate within a few days after the discontinuance of quinine therapy, in others, fibrillation recurs after weeks or months and there are only relatively few cases in which it does not recur. In hyperthyroidism defibrillation has scarcely any value, since the tendency to fibrillation is so great that without successful treatment of the underlying malady sinus rhythm lasts only a short time. The same holds for many cases of mitral stenosis, coronary sclerosis, etc. To this may be added the fact that in most cases the return to sinus rhythm brings no decisive improvement, and the patient usually does not perceive the trans formation at all.

Quinidine treatment of fibrillation, aimed at the restoration of sinus rhythm, is *never unconditionally indicated or absolutely necessary* since the same success can be obtained with digitalis. For this reason one must always ask whether or not there is justification for subjecting the patient to danger without the assurance of more certain favourable results. Indeed defibrillation is always asso ciated with a certain amount of danger, even when the contra indications mentioned above are observed.

Accordingly quinidine therapy is advised only when there are definite reasons for believing that with due regard for all the cir

cumstances the patient's condition can be improved with certainty Such cases, to be sure, are not common A few examples may be cited A subtotal thyroidectomy has been undertaken in a patient *with severe hyperthyroidism and auricular fibrillation*, but without marked enlargement of the heart After the operation all the signs of hyperthyroidism vanish but the fibrillation persists and compels continuous digitalization In such a patient an attempt to defibrillate with quinidine may be made, since thereby a complete *restoration of health* may be achieved *Another instance* auricular fibrillation appears during the course of pneumonia The pneumonia subsides but the fibrillation persists In such circumstances one has not only the right but the duty, to attempt to restore sinus rhythm Again a patient with mild mitral stenosis without marked *dilatation of the left auricle begins to fibrillate In this instance the* fibrillation may be harmful, because it paralyzes the auricles the contractions of which materially assist in maintaining compensation indeed auricular activity helps by forcing blood into the ventricle In these cases the defibrillation is usually not permanent as it is in *the two examples cited earlier* However if it is possible for only six months to maintain full compensation without digitalis this has its advantages

Prior to quinidine treatment it is advisable to digitalize the patient until there is a ventricular rate of 70–80 Then digitalis may be discontinued and quinidine started During quinidine therapy strict rest in bed must be maintained, since the patient requires supervision the treatment should on principle be carried out only in a hospital

At first a single dose of 0 25 gm ($3\frac{3}{4}$ grs) of *quinidine is given by* mouth, the more powerfully acting quinidine is superior to quinine If by the next morning no manifestations of hypersensitivity have appeared, if vertigo and tinnitus diarrhœa and erythema are absent, then the actual treatment is started Since the manifestations of *hypersensitivity are not rare and since occasionally they may attain* an alarming degree the administration of a test dose is unconditionally advised

The procedure employed is as follows

1st day	$3 \times 0\ 25$ gm	($3\frac{3}{4}$ grs) quinidine
2nd day	$4 \times 0\ 25$ gm	
3rd day	$5 \times 0\ 25$ gm	
4th day	$6 \times 0\ 25$ gm	(or $3 \times 0\ 50$ gm)
5th day	$3 \times 0\ 50$ gm	
6th day	$3 \times 0\ 50$ gm	
7th day	$3 \times 0\ 50$ gm	

In other words, one begins with a dose of 3×0.25 gm per diem and increases the dose by 0.25 gm each day In feeble patients in young individuals and in delicate women it is well not to exceed 1.5 gm a day Only in robust adults with a good myocardium do the authors increase the daily dose to 2 gm Naturally larger doses are given only when the smaller amounts are tolerated, if untoward effects of quinidine appear then treatment is interrupted If the fibrillation ceases on one of the seven days (it may stop on the first or second) the initial dose of 0.25 gm three times a day is continued for a few days and then stopped If examination on the eighth day shows that the fibrillation is still present the treatment is interrupted and one must remain resigned to digitalis therapy Only when the dose is increased cautiously and gradually and quinidine is not prescribed in too large doses and not for too long a time are undesirable complications avoided Since quinidine never *must* be given it is better to avoid it if there is any doubt about its being necessary or tolerated

During the administration of quinidine the pulse rate often increases considerably this is particularly striking as the heart is usually quite slow from the preceding digitalis therapy This untoward action which not rarely disquiets the physician in charge has its origin in the property of quinidine of producing a peripheral paralysis of the vagus As after an injection of atropine so after quinidine no depressant action on the heart may occur experimentally when faradic stimulation is applied to the trunk of the vagus If a corresponding dose of quinidine (0.2 0.3 gm) is injected intravenously into a healthy person its direct paralyzing action on the vagus may produce a considerable tachycardia only when larger doses are administered does a direct paralyzing action on the specific fibres and thereby a slowing of the rate become evident In cases of auricular fibrillation this effect of quinidine causes the disappearance of the ventricular slowing previously induced by digitalis This acceleration of rate is by no means a dangerous sign

Often digitalis is not administered alone in fibrillation but is combined with quinidine The antagonistic action on the vagus of the two drugs mentioned above makes this combination seem inappropriate upon theoretical grounds Clinical experience likewise is not favourable, as a matter of fact the most essential component of digitalis action in auricular fibrillation the increase in vagal tone is abolished by quinidine

Clinical Remarks on Auricular Flutter

Auricular flutter often regarded as a preliminary stage to

fibrillation appears under the same conditions as fibrillation But it is by no means a necessary preliminary stage it is much rarer than fibrillation

The clinical diagnosis is not always easy If a continuous 4 1 or 5 1 block is present the ventricle contracts regularly with a rate corresponding to the normal (Figs 108b 111c) In cases of this kind the regular very fast venous waves produced by the fluttering auricle only rarely are seen in the neck although when evident they *facilitate the diagnosis Only in slighter grades of block (2 1 3 1)* does a tachycardia exist at first to be sure it cannot be differentiated from a simple acceleration of cardiac action Very often the block is irregular so that at clinical examination all possible allorhythmias and even the complete arrhythmia of auricular fibrillation are presumed to be present

With some scrutiny the diagnosis of auricular flutter is usually possible without an electrocardiogram if the existence of this *disturbance is considered The auricular rate in flutter usually* ranges around 300 but it may lie between 200 and 400 Since in a majority of cases a 2 1 or 3 1 block exists the ventricle contracts between 100 and 160 times a minute For this reason it should be made a rule that the possibility of the existence of flutter should always be given consideration when such ventricular rates are present The differentiation of flutter from simple acceleration then becomes simple In sinus tachycardia even sitting up and *especially standing up or slowly walking around the room causes* a slight increase in rate on the contrary in flutter the ventricular rate remains unaffected under these conditions since change in position or slight movement does not influence either auricular activity or conduction to the ventricles But if greater exertion is permitted for example the knees are bent fifteen to twenty times or when this cannot be performed a few drops of amyl nitrite are inhaled from a cloth for twenty seconds in sinus tachycardias *a simple acceleration appears and then gradually subsides on the* contrary, in auricular flutter the lowering of vagal tone and the increase of sympathetic tone lead to better conduction *i e* to ' unblocking the flutter rate in the auricle is not changed. Thereby a 2 1 block may appear in place of a 3 1 in this instance for example *a ventricular rate of 100 will increase to 150* or a very fast tachycardia of 300 may appear in other words there is a simple mathematical ratio between the rate before and after the exercise or before and after the *inhalation of amyl nitrite* Some minutes after the exercise the rate returns to its original level, at

times this occurs suddenly but more frequently the change follows a brief arrhythmia

Fig 124a shows auricular flutter with a 2 1 block, after bending the knees twenty five times, every stimulus was conducted

a b

FIG 124 Auricular flutter during rest with 2 1 block (a) after exercise a full rhythm appears (b)

(Fig 124b), the ventricular rate became exactly doubled and increased from 107 to 214

In Fig 125 on superficial examination one might think that a normal sinus rhythm was present since normal P waves are followed by initial and terminal deflections which are normal in shape Measurement with dividers however shows that auricular flutter with 3 1 block exists again one F wave is isolated one is concealed by the initial and another by the terminal deflection But sinus rhythm may occasionally occur in which the interval between the waves is accidentally equal Since the diagnosis could not be made from Lead II, which is reproduced nor from the other leads, the electrocardiogram was recorded after the patient had climbed

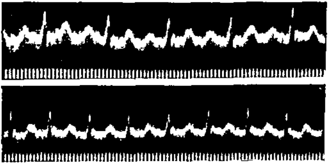

FIG 125 Above auricular flutter with 3 1 block below (after exercise) flutter with 2 1 block

stairs If a simple sinus rhythm existed cardiac acceleration alone would appear after the exercise But in this case as is evident from Fig 125, the ventricular rate increased exactly 50 per cent In place of a 3 1 block a 2 1 block developed The auricular rate was 272 The ventricular rate with 3 1 block amounted to 90,

with 2 : 1 block to 136; that is, after exercise it increased by exactly 45 beats

In auricular flutter not rarely an irregular block exists so that the examining physician makes a diagnosis of auricular fibrillation Here also an exercise test or the inhalation of amyl nitrite facilitates the diagnostic decision *In fibrillation the ventricular irregularity is augmented*; in flutter a continuous 2 : 1 or 3 : 1 block occurs in place of a varying 2 : 1 and 3 : 1 block

The subjective complaints of patients with auricular flutter vary considerably With high degrees of block and for this reason low ventricular rates the patient may feel fairly well and also does not require treatment; *cases of this kind are not uncommon* Some must merely refrain from heavy work; otherwise the "unblocking" leads to a very fast tachycardia and marked palpitation But it should be evident that flutter with a 2 : 1 block and a corresponding ventricular tachycardia causes symptoms and that these appear the more rapidly and severely the higher the rate of the ventricles For this reason everything which was discussed in the chapter dealing with fibrillation might be repeated here; it was stated at the time that it is not the auricular anomaly but the ventricular rate which is significant

The treatment of auricular flutter is more difficult than that of fibrillation In the latter by small doses of digitalis causing a slight increase of vagal tone the weak auricular stimuli can be prevented from reaching the ventricles But in flutter the auricles contract much less frequently and therefore more powerfully; the stimuli which reach the A–V system are stronger and are not blocked on their way to the ventricle by small doses of digitalis No slowing of the ventricle is obtained after the usual amounts of digitalis If larger doses are administered they may provide an effect, but larger amounts of digitalis cannot be given over a long period of time because the most unpleasant untoward effects soon appear For this reason digitalis therapy fails if it is carried out in the usual manner

However animal experimentation has shown that auricular flutter can immediately be converted into auricular fibrillation when the vagus in the neck is stimulated by a faradic current It is a simple experiment which is successful every time A series of hypotheses have been devised to explain this action but all of them remain unproven Nevertheless this experimental observation is utilized clinically in that patients with auricular flutter are treated with very large doses of digitalis (corresponding, for example,

to 6×0.1 gm (9 grs) a day) Thus an attempt is made to produce a sharp increase of vagal tone in order to obtain the same result as was produced in the animal by electrical stimulation of the vagus this procedure succeeds in a fair percentage of the cases In many patients digitalis therapy must be carried to the point of vomiting Digitaline Nativelle (10–15 drops three times a day) is most effective because it is very toxic and cumulates readily

When fibrillation appears treatment becomes much easier at that stage much smaller doses of digitalis are required to prevent the recurrence of flutter and to maintain a slow ventricular rate To be sure there are cases in which a slight reduction of the dose of digitalis permits the flutter to recur

If digitalis has proven ineffective the patient continuing to have flutter while taking large doses of digitalis quinidine is tried in the same way as was mentioned in regard to fibrillation The danger of embolism is much less because in flutter the auricles contract more powerfully and thrombosis rarely occurs

But unfortunately there are cases in which neither digitalis nor quinidine are successful and flutter persists without change In these cases there is no other therapeutic possibility which can be applied with expectation of success After some months have elapsed one may repeat both agents at times with more success

As with fibrillation there always remains the hope that some day the flutter will suddenly vanish Occasionally the flutter is converted into fibrillation for unknown reasons and the situation rapidly improves

Clinical Remarks on Ventricular Flutter and Fibrillation

The clinical aspects of ventricular flutter will not be discussed at this juncture because all the essential phases of the subject are considered under the heading of ventricular tachycardia Just as it is difficult to draw a sharp limit between paroxysmal auricular tachycardia and auricular flutter and just as one cannot *always* say which of these two conditions is present so many cases of paroxysmal ventricular tachycardia are designated as ventricular flutter Indisputable ventricular flutter is very rare usually a very fast ventricular tachycardia is present

If in ventricular flutter even with a rate of 300 the circulation is profoundly disturbed then the onset of ventricular fibrillation will signify immediate standstill of the circulation and in most cases death Ventricular fibrillation is perhaps the most common cause of death in disease of the coronary arteries It occurs suddenly

only rarely is it foreshadowed by polytopic extrasystoles In rare cases ventricular fibrillation appears for only a couple of seconds or minutes, recovery or the return to regular cardiac action however, is exceptional In the chapter on Stokes Adams syndrome some additional remarks on this subject will be found (p, 300)

It is interesting that most attacks of transient ventricular fibrillation are observed in patients who for some reason have received an intravenous injection of quinidine (Schwartz and Jezer) Often this happens in cases with a disturbance of auriculo ventricular conduction who show extrasystoles tachycardias and ventricular fibrillation *This action is paradoxical because quinidine normally* depresses stimulus formation in the heart and removes fibrillation However in experiments it has been observed after aconitine was employed (Scherf and Siedeck)

The fact that the same agent at one time may paralyze and at another time may stimulate the formation of extrasystoles holds not only for quinidine but also for numerous other substances *In this connection it may be recalled that digitalis abolishes the* extrasystoles (p 203) which occur in healthy individuals but under certain conditions precipitates them in diseased hearts (p 177) if digitalis therapy is continued it may produce even ventricular fibrillation The same holds for epinephrine potassium calcium etc Actually it can be affirmed as a general rule that according to the state of the receptive organ (in this instance the specific fibres *of the heart) the same stimulus may act in entirely opposite ways*

Ventricular fibrillation also may appear when digitalis or strophanthin is continued despite the presence of digitalis extrasystoles

No treatment is of any avail In animal experiments one can immediately terminate the fibrillation by an intravenous or better an intracardiac injection of a 1 per cent solution of KCl If then one washes the solution of potassium chloride out (say in a Langendorff heart preparation) and perfuses the heart with a fresh nutrient solution it may contract powerfully for a long time In hearts *in situ* one succeeds only rarely in expressing the potassium by long continued careful massage and reviving the heart For obvious reasons this method is not applicable in man But numerous cases have been observed in which the attacks of ventricular fibril lation ceased spontaneously and gave way to sinus rhythm (case of *Fig 120)*

Origin of Flutter and Fibrillation

The mode of origin of auricular flutter and fibrillation is only imperfectly understood Although scarcely any other pathological

condition can experimentally be produced with such ease and regularity our understanding of the clinical condition is still in the stage of theories and attempts at explanation

Among the latter the authors discuss only two all the others have been discarded as knowledge of this disturbance of rhythm has become more extensive by an exact study of the clinical forms of this phenomenon and on the basis of very impressive animal experiments

According to the view of Engelmann Hering Winterberg and others in flutter and fibrillation the underlying disturbance is *a very fast rate of stimulus formation* which originates in multiple local sites of activation this may take place in the most diverse areas of cardiac muscle Subsequent animal experiments led Rothberger and Winterberg to the conception of *one* centre of stimulus formation this has become plausible by virtue of the clinical observation that auricular and ventricular fibrillation may end abruptly usually without an intermediate alteration of the electrocardiographic picture If several foci were active it would seem improbable that they should always become inactive simultaneously In this conception fibrillation would constitute the utmost disturbance of stimulus formation The site of abnormal stimulus formation was formerly located in the specific tissue of the sinus or A–V node but it seems that fibrillation can have its origin wherever specific fibres are present

Repeatedly scepticism has been expressed whether or not so rapid a formation of stimuli would be possible for a long period frequently throughout life In this connection it may be said that normal cardiac rates of 300 or more occur in many mammals, in many birds a cardiac rate is normally found which exceeds that occurring in human fibrillation

According to the *circus movement theory* which is supported by the investigations of Mayer Mines Garrey and Lewis auricular flutter and fibrillation are ascribed to a very rapid circulation of one or perhaps of several waves of excitation in one or more closed muscle paths of which many are present in the auricle

This explanation becomes more comprehensible if the basic experiments of Mayer and Mines are recalled A tortoise heart may be used as an example The heart of this animal has only one ventricle and the apex and base can be removed so that only a muscle ring remains which one can slide over the finger (Fig 126) If this muscle ring is at any optional place stimulated by an induction shock, a wave of excitation spreads over it in both directions and

stops when the waves meet But if one stimulates at A and simultaneously, by means of a clamp, compresses the muscle next to the *site of stimulation, the wave of excitation can go in but one direction* namely, that which is not obstructed by the clamp When the wave of excitation returns to its point of origin, and if this is again excitable (the clamp having been removed in the meantime), the wave *again can circulate over the path* Many repetitions of this fundamental experiment have shown that an excitation which is compelled to move in *one* direction in a closed circuit can circulate for hours The wave of excitation passes around *several hundred times a minute the more rapidly* the smaller the ring and the better the state of the muscle fibres

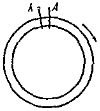

FIG 126 Schematic drawing for the explanation of the circus movement theory

Even the first investigators who performed this experiment raised the question of whether or not *auricular fibrillation develops through the circulation of such a* wave It is known that not only closed musculature exists in the auricle but also a well developed network of muscle bundles (Fig 3) The "central or mother wave" (Lewis) could circulate in such a ring and the waves of excitation, radiating centrifugally from it, could excite the auricle and ventricle

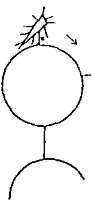

FIG 127 Schematic drawing of the circus movement theory in auricular fibrillation

But how does this circulation occur in man, what is the substitute for the clamp, what takes the place of the electrical stimulus of the experiment? As a matter of fact, as the following discussion shows, there is actually the possibility of imitating the clamp and the electrical stimulus in the heart Fig 127 shows the sinus node and its junctional tissues One of the fibres leads to excitation of the muscle ring A from there it passes through the *auricular muscle paths and the A–V system to the ventricle, V It* may be assumed that a wave of excitation passes over the auricle and immediately thereafter an auricular extrasystole originates at x In regard to auricular extrasystoles it has been stated earlier that owing to their prematurity they encounter tissue which has not *recovered as yet, and for this reason they are often retarded in the*

bundle of His or are not even conducted to the ventricle Thus, it is possible that the auricular extrasystole occurring early, finds the auricular tissue so incompletely recovered from the conduction of the preceding normal stimulus that it is transmitted in the muscle ring A only in the direction of the arrow Through this unidirectional movement the chief condition for the circus movement is given and the mechanism is actually *conceivable* However, it is still *unproven*

Numerous objections to this interpretation can be raised, there are clinical and experimental observations which are susceptible to different more simple and perhaps better interpretations (Rothberger Winterberg Scherf) It is also difficult to understand circus movement in ventricular fibrillation and flutter, that is in states whose electrocardiographic picture closely resembles auricular flutter and fibrillation There is no closed muscle ring in the ventricle, but simply a syncytium of muscle fibres It is also difficult to understand according to the circus movement theory why continuous fibrillation appears only in muscle bundles which contain specific fibres (Nomura), but otherwise is always absent The auricular appendix isolated from the auricle by clamping ceases to fibrillate (Garrey)

Lewis endeavoured to secure proof of circus movement by two main methods Utilizing a double string galvanometer, the electrogram of the fibrillating auricle of a dog was registered by a direct lead with Lead II simultaneously recorded By moving the electrode to different parts of the auricle it could be decided when the auricular area under investigation at the moment was excited and by sampling all of the parts of the auricles it could be demonstrated that the central or mother wave utilized the same circular pathway In practically all of the cases examined (one exception) it went up or down the tænia terminalis, embraced the superior vena cava above and the inferior vena cava below, the circle was closed posteriorly But for technical reasons this final sector of the path could not be investigated and Lewis could only assume its utilization by means of calculations Nevertheless, the final and conclusive proof was not available

Likewise an objection is tenable (Rothberger) against a second experimental arrangement which is employed to prove the correctness of the circus movement theory Lewis recorded the electrocardiogram of a patient with auricular flutter simultaneously in three leads and calculated the direction of the spread of excitation from the form of the auricular waves He concluded that it circles around 360 degrees But the spread of excitation of the central

wave in a small band of muscle cannot be deduced from the form of the F wave the activation of this path scarcely produces a deflection in the electrocardiogram the auricular wave depends primarily upon the activation of both auricles which according to the circus movement theory proceeds through centrifugal waves which radiate out in all directions from the central wave

The following experimental arrangement also speaks against the *circus movement theory* *According to Lewis in flutter and fibrillation* the central wave practically always employs a pathway which embraces the tænia terminalis accordingly in animal experiments performed in dogs an attempt was made to interrupt this path by a broad transverse ligature across the tænia terminalis In sixteen out of seventeen experiments the flutter remained unchanged and continued without modification of the electrocardiogram in a single experiment it immediately stopped this cessation does not prove much because in experimental auricular flutter any contact of the auricle now and then leads to a restoration of sinus rhythm In a second series of experiments a striking observation was made namely *that the form of the flutter waves had remarkable similarity* to the P waves at the time If certain paths were tied off for example the interauricular bundle described by Bachmann which decidedly modifies the form of the P waves then the form of the P waves changed and the form of the flutter waves now approximated the form of the P waves

In these experiments in order to study the form of the flutter waves a heart block was produced by cross section of both bundles In this way the tachycardia of the auricles was prevented from reaching the ventricles Fig 128 shows sections of tracings from three experiments They show the vast similarity between the form of the P waves and the form of the flutter waves If the P waves are negative then the flutter waves are negative if the P waves are splintered the flutter waves are splintered also

This identity of the form of the P waves and of the flutter waves is difficult to understand according to the circus movement theory particularly when it is recalled that according to Lewis the circus wave is supposed to utilize the same pathway in practically every animal experiment regardless of the site of the stimulation which caused it

Another argument against the circus movement theory is the *following observation* Several cases (Cookson and Clark Kennedy Parsonnet and Parent) have been observed in which the auricular rate in auricular flutter suddenly halved Thus in one case it fell from 412 exactly to 206 and in another with an auricular rate of

Fig 122. Dog experiment Striking similarity between the P waves and the F waves in experimental flutter

460 (as measurement shows, although the authors report a different figure), exactly to 230 Cookson and Kennedy are convinced as to the correctness of the circus movement theory, so that they propose quite implausible suggestions in order to avoid recourse to any other explanation Among others they discuss the possibility that the central wave suddenly employs a path in the auricle which is exactly twice as long so that the rate becomes one half In our opinion it is much more reasonable to assume the following interpretation *412 stimuli were continuously formed and transiently the auricle* responded to only every second one (Camp and Scherf)

In an experiment it *is not* difficult to produce fibrillation of either the right or the left auricle whereby the unaffected chamber continues to beat in a slow rhythm (Scherf and Siedeck)

On the basis of these observations we believe that the existence of a circus movement in auricular flutter and fibrillation is still not completely established In the light of our present knowledge, circus movement as the cause of flutter and fibrillation is just as unproven as the theory of rapid formation of stimuli Some observations suggest, however, that flutter and fibrillation are entirely different from other types of rapid stimulus formation in a centre, for example, in paroxysmal tachycardias This is evident from the fact that auricular flutter and fibrillation cannot be arrested by vagus irritation as is the case in the tachycardias, rather they are aggravated and prolonged Whereas stimulation of the vagus abolishes paroxysmal auricular tachycardia, it transforms auricular flutter into auricular fibrillation

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THE TACHYCARDIAS

The tachycardias are appropriately classified as sinus tachycardias and paroxysmal tachycardias The latter group is subdivided into three types

Sinus Tachycardia

In sinus tachycardia the most common form of the tachycardias, there is simply an acceleration of the normal sinus rhythm It

a

b

c

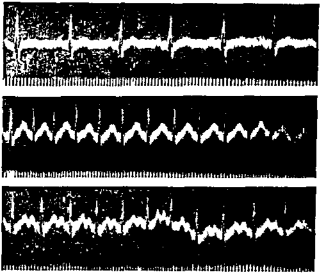


FIG 129 (Lead II) Above an electrocardiogram during rest (a) in the middle sinus tachycardia after five minutes of standing (b) below after an injection of atropine (c)

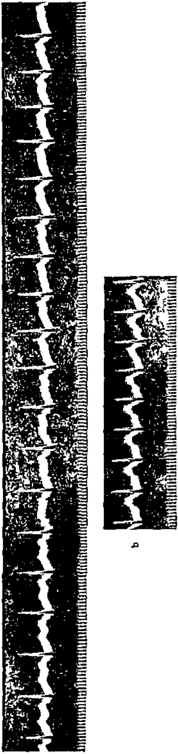
occurs after rapid walking or from other forms of physical exertion, it is found in febrile patients in whom the rate increases about 8–10 beats for each degree of temperature above normal It develops in poisoning with atropine epinephrine etc, it is a common symptom of hyperthyroidism of cardiac neuroses and in patients with unstable vegetative nervous system and irritable hearts There are infectious diseases and apparently harmless chills in which the patient may exhibit an accelerated heart rate for weeks after all other morbid symptoms and signs have disappeared

Sinus tachycardia does not always represent a harmless acceleration of the pulse To be sure, the cardiac rate usually ranges between 100–120 but may rise to 150–180, and even reach 200 If the minute rate exceeds 200, sinus tachycardia usually may be excluded, paroxysmal tachycardia being present It is comprehensible that with these high rates circulatory disturbances may appear

In sinus tachycardia there is a characteristic and distinct acceleration of the rate upon the slightest physical exertion, *such as the change of position of* the patient Thus, in a case of hyperthyroidism or of a cardiac neurosis a rate of 120 per minute may be found during quiet rest in bed but upon standing the rate may rise as high as 180 This remarkable instability of the pulse and its susceptibility to effort and excitement is characteristic of sinus tachycardia and decidedly reduces the functional capacity of the individual

The electrocardiogram of Fig 129 was *recorded from a case of* cardiac neurosis The upper tracing (Fig 129a) shows a sinus tachycardia with a rate of 140

Fig 129b shows a marked cardiac acceleration after five *minutes of standing* The minute rate is 187 By virtue of the tachycardia the diastole has be



a b

Fig 130a and b (Lead II) Sinus tachycardia provoked by inhalation of amyl nitrite with increasing shortness of diastole the P waves become hidden in the preceding T waves

come so short that the P waves are hidden in the T waves of the preceding beat

From Fig 129c it is evident that the cardiac acceleration following a subcutaneous injection of ½ mg (1/120 gr) of atropine amounts to only 162

The electrocardiogram of sinus tachycardia shows the picture of an accelerated cardiac action with normal origin of excitation The ventricular complexes have the form otherwise characteristic of the individual when the tachycardia affects a healthy person they are normal in form when in addition to the sinus tachycardia disturbances of intraventricular conduction exist they may appear abnormal Differential diagnostic difficulties arise when the rate is fast and the ventricular diastoles become increasingly shorter so that the P waves to an increasing degree are merged with the T waves of the preceding beats and are finally concealed in them

Accordingly in Fig 130a one sees a sinus tachycardia which becomes increasingly faster produced in a healthy individual by the inhalation of amyl nitrite The T waves more and more merge with the P waves until finally the two waves cannot be distinguished from each other (see Fig 130b and also Fig 129b) Confusion with other forms of tachycardia is especially possible when a patient with a fully developed sinus tachycardia is examined Reference to this will be made later

Classification of Paroxysmal Tachycardias

Paroxysmal tachycardias are differentiated from sinus tachycardias by their sudden beginning and sudden end they begin like lightning as suddenly as a light is turned on or off and end just as abruptly At times the sudden beginning of cardiac palpitation is also mentioned by patients with sinus tachycardia One may recall the palpitation of fright But sinus tachycardias always decline slowly in contrast to the sudden cessation of the paroxysmal type Sinus tachycardia in hyperthyroidism in cardiac neurosis or after infectious diseases vanishes only after the lapse of weeks although a tachycardia after exertion may vanish in the course of a few minutes

Three principal forms of paroxysmal tachycardia are distinguished (1) paroxysmal flutter and paroxysmal fibrillation (2) paroxysmal ventricular tachycardia and (3) paroxysmal auricular tachycardia The other rare forms (atrioventricular tachycardia) are considered elsewhere

Paroxysmal Flutter and Fibrillation

These are very common conditions. The disturbances considered in the preceding chapter do not always last for a long time nor do they always persist for a long period to vanish spontaneously or after therapy. *Frequently they remain for only a short time for seconds minutes or hours.* Since *these attacks begin suddenly and end suddenly* and since they are usually accompanied by fast ventricular rates they may be called paroxysmal tachycardias. The complaints of the patient and the effects on the circulation are the same as in other forms of paroxysmal tachycardia.

If the patient states that sudden attacks of palpitation are accompanied by irregular cardiac action the diagnosis of auricular

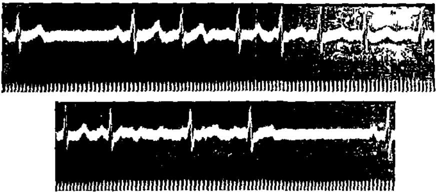


Fig 131 Beginning and end of an attack of paroxysmal auricular fibrillation

fibrillation may be presumed from the history. Attacks of this kind are particularly common in older patients with coronary sclerosis. However they are also found in young individuals who but for the attacks remain for years or decades without symptoms or signs of organic heart disease.

Fig 131 shows the sudden beginning and the sudden end of an attack of paroxysmal auricular fibrillation. The attack begins with an auricular extrasystole in the T wave of the second normal beat (see p 235). The P waves vanish and the F waves irregular in form are visible in the longer diastoles. The appearance of the initial deflections and even more distinctly the form of the T waves is continually altered by the summation with the F waves.

Fig 132 shows three brief attacks of paroxysmal fibrillation and flutter lasting only a few seconds. Owing to their premature

Fig 133 Very short and frequent attacks of paroxysmal auricular fibrillation and flutter

appearance a few auricular stimuli are conducted to the ventricle in an entirely abnormal manner (aberrant conduction)

A short portion of a tracing may be seen in Fig 133 it was recorded *during* an attack of paroxysmal fibrillation The ventricle contracts so rapidly (approximately 180 times a minute) that the difference in the length of the individual diastoles (the arrhythmia) is very slight Owing to the shortness of the diastoles F waves are not visible The variable form of the ventricular complex (chiefly of the T waves) here also proves that they summate irregularly with the F waves and that auricular fibrillation exists

If paroxysmal fibrillation is accompanied by a low ventricular rate it requires no treatment Often it is not even noted by the patient

Paroxysmal Auricular and Ventricular Tachycardia

In discussing extrasystoles it was emphasized that at times they are not isolated but appear in groups that is they are multiple as a rule appearing at short intervals after each other (Fig 96) If not one or five or ten but say 500 or a great many more extrasystoles appear in an uninterrupted serie the condition is called paroxysmal tachycardia viz auricular tachycardia when auricular extra systoles and ventricular tachycardia when ven tricular extrasystoles follow each other

In other words paroxysmal auricular and ven tricular tachycardias consist of nothing more than a long chain of extrasystoles When one speaks in general terms of paroxysmal tachycardia usually reference is made to one of these two forms

The Electrocardiogram of Paroxysmal Ventricular Tachycardia

The diagnosis of paroxysmal ventricular tachy cardia by means of the electrocardiogram is usually

relatively easy. One sees a chain of abnormal initial and terminal deflections which are not preceded by P waves.

In Fig 134a the sinus beats entirely normal in form show a conduction time prolonged to 0.26 second. Then two single extrasystoles appear. Between the QRS complex and the T wave of the

Fig 133 (Lead II) Auricular fibrillation with very fast ventricular rate

extrasystoles the blocked P wave of a sinus beat may be seen distinctly. At the end of the curve in Fig 134 paroxysmal ventricular tachycardia appears; actually a chain of ventricular extrasystoles is seen. In Fig 134b at a few places during the tachycardia the P waves can be recognized which indicate that the auricles

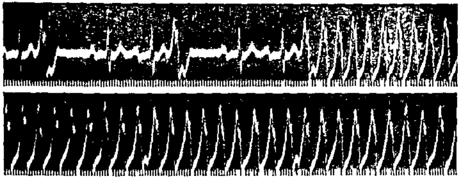


Fig 134 Isolated ventricular extrasystoles and the beginning of an attack of paroxysmal ventricular tachycardia. The conduction time of the sinus beats is prolonged to 0.26 second

continue to beat in sinus rhythm. Thus it is probable that the R wave of the seventh extrasystole and the isoelectric line between the ninth and tenth conceal a P wave.

The form of the QRS complex varies in each individual case according to the point of origin of the tachycardia and the spread of excitation occasioned by it. As with isolated ventricular extrasystoles, extrasystoles of ventricular tachycardia, as a rule, are not

conducted back to the auricle, so that in ventricular tachycardia actually an auriculo ventricular block is always present The auricles contract regularly in a normal way under the control of the sinus node, yet one can find P waves only rarely at various places in the electrocardiogram, since being small they are lost in the QRS complexes and T waves of the extrasystoles

The ventricular rate in paroxysmal ventricular tachycardia varies It may exceed the existing normal rhythm only a little but there are also minute rates of 250 and more In such cases ventricular flutter is very often said to be present (p. 215) Ventricular tachycardias are usually regular It is often asserted that they show irregularities which are demonstrable by auscultation (Strong and Levine) But more recent investigations have failed to corroborate these findings (MacKinnon) Irregularity occurs only in some special types of ventricular tachycardia (Fig 137) Figs 134 and 135 show the great regularity of typical cases It has also been stated that the irregularity may be found on auscultation This is usually impossible, because the fast rate prevents marked differences in the length of diastole But changes in the intraventricular conduction (Fig 137), and occasional summation with auricular contractions may change the intensity of the heart sounds

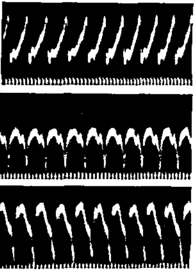


Fig 135 Paroxysmal ventricular tachycardia

Fig 135 shows short sections of a tracing in all leads from a case of paroxysmal ventricular tachycardia Again abnormal ventricular complexes in very rapid sequence are seen The minute rate amounts to 300 The diagnosis of ventricular tachycardia is substantiated by observation after the end of the attack, when in this case one found an entirely normal electrocardiogram which was interrupted by single ventricular extrasystoles, these, as Fig 134 shows exhibited the same appearance as the extrasystoles which composed the tachycardia

Just as in rare cases *single* extrasystoles may develop above the bifurcation of the bundle and then spread normally in the ventricle,

so at times in a ventricular tachycardia with a similar high origin of the stimulus, the QRS-complexes may exhibit a normal form. The distinction between auricular tachycardia and atrioventricular tachycardia is then difficult (see p. 171). On the other hand, occasionally the presence of ventricular tachycardia may be wrongly diagnosed, since the ventricular beats in an auricular tachycardia may appear abnormal owing to fatigue of the conduction path and aberrant conduction in the ventricle or to the development of an auricular tachycardia in a patient with disturbance of intraventricular conduction.

Fig. 136a shows auricular bigeminy. The tracing was obtained from a patient with a mitral valve lesion and myocardial damage (myo-pericarditis). The P-waves of the normal beats are widened

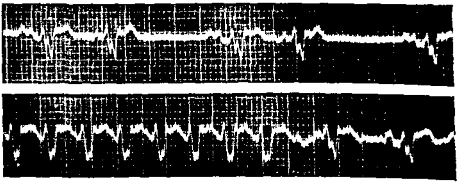


FIG. 136. Auricular bigeminy (a) and the end of an attack of paroxysmal auricular tachycardia (b) in a case of disturbance of intraventricular conduction.

and bifid. An auricular extrasystole appears after each normal beat. In the lower tracing (Fig. 136b) the spontaneous end of a paroxysmal auricular tachycardia is seen. Since the auricular extrasystoles then followed each other so rapidly, the P-waves are no longer distinct. If the electrocardiogram had been taken only in the attack, one would have been inclined to assume the presence of a ventricular tachycardia because of the rapid sequence of the abnormal ventricular complexes.

In discussing ventricular extrasystoles and bigeminies, the important fact was emphasized that variation in form of the extrasystoles, and a constant change in their appearance, favours the diagnosis of myocardial disease ; often it is not only the myocarditis, but digitalis therapy which in a diseased heart brings about this kind of extrasystoles. In some cases (through the continuation of

digitalis therapy), these extrasystoles multiply so that one finds a chain of extrasystoles with constantly changing appearance At times one also sees an alternation of two forms These tachycardias are also called terminal tachycardias (Gallavardin) since they often appear just before death If the tachycardia has been produced by digitalis therapy, it may vanish with the timely interruption of therapy and by the administration of quinidine

In Fig 137 there is a typical example of a ventricular tachycardia of this kind It appeared in a patient with a severely decompensated coronary lesion who had been treated with small doses of digitalis (3 × 0 05 gm daily) Initially the ventricular complexes are similar in shape but in contrast to other ventricular tachycardias, they appear at irregular intervals After the fifth extrasystole, ventricular complexes of various shapes are seen and soon the two forms of extrasystoles continually alternate with each other

The alternation may be absolutely regular for hours and days Occasionally there is also an alternation of a longer and a shorter diastole but frequently the rhythm is very regular If the rhythm and form of the ventricular contractions change continuously, the condition is called " anarchie ventriculaire (Clerc and Levy)

The lesion is best explained by disturbances of intraventricular conduction (Scherf)

Fig 137 Paroxysmal ventricular tachycardia with changing in part alternating forms of extrasystoles after digitalis

The Electrocardiogram of Paroxysmal Auricular Tachycardia

Paroxysmal auricular tachycardias also are composed of a chain of extrasystoles As with single extrasystoles, the appearance of the P wave may vary Inverted P waves are very common but they may also be positive, split or broadened Here also there are

tachycardias which are only a little faster than the sinus rhythm of the case concerned and there are tachycardias with very fast rates As in flutter disturbances of conduction between the auricle and ventricle also may appear in fast auricular tachycardias so that as a result of fatigue of the conduction system the beats are conducted aberrantly to the ventricle in such cases wide abnormal QRS complexes appear These tachycardias may then give the impression that they were ventricular in origin As with single

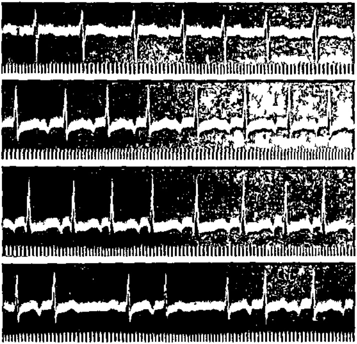


Fig 138a–d Paroxysmal auricular tachycardia and in (d) an auricular *bigeminy and the beginning of a new attack*

extrasystoles one must always try to determine whether or not the ventricular beats are preceded by P waves

The three upper tracings Fig 138 are the three standard leads from a case of paroxysmal auricular tachycardia The rate amounts to only 127 In this case it happens that there are inverted P waves in all leads which are immediately apparent after the T waves of the *preceding beat* In the lower tracing (Fig 138d) one perceives a single auricular extrasystole after each of the first two normal beats Following the third normal contraction a new attack of tachycardia begins The electrocardiogram was obtained from a patient with a mitral valve lesion therefore a *dextrocardiogram was obtained*

In Fig 139a the beginning and in Fig 139b the end of an attack of paroxysmal auricular tachycardia are shown A considerable section of the tracing has been removed between Fig 139a and b Since the rate amounts to approximately 200, the P waves are hidden in the T waves of the preceding beats Before and after the tachy

a

b

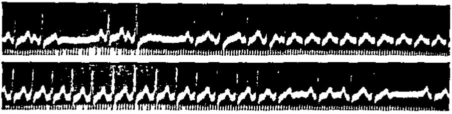


Fig 139a and b Beginning and end of an attack of paroxysmal auricular tachycardia Before and after the attack auricular extrasystoles appear which are conducted aberrantly to the ventricle

cardia there exists an auricular bigeminy with aberrant conduction of the auricular extrasystoles which appear very premature

In Fig 140 there is a paroxysmal tachycardia with a rate of 125 beats per minute The P waves of the extrasystoles are hidden in the T waves of the preceding beats In the first beats a prolonged conduction time exists then a transient 2 1 block and dropped beats appear These disturbances of conduction between auricle and ventricle appeared in the course of digitalis therapy On the

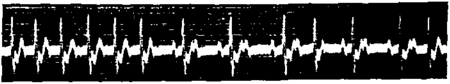


Fig 140 (Lead II) Paroxysmal auricular tachycardia with conduction disturbance to the ventricle

next day the tachycardia vanished and was replaced by normal sinus rhythm

Fig 141 shows portions of tracings from four different cases of paroxysmal tachycardia The first and the second tracings are examples of a paroxysmal auricular tachycardia, the third and fourth cases are auricular flutter with 2 1 block The flutter is easily recognized only in the fourth tracing since one is able to detect the beginning of an F wave directly before the ventricular complexes Fig 141c however closely resembles the tracings of

Fig 141a and b A slight depression of the S T segments in Fig 141c may be the result of the tachycardia but might also be caused by myocardial injury, in the latter instance this depression would also have to be present with the normal sinus beats after the cessation of the tachycardia

A very common difficulty in diagnosis is encountered when the paroxysmal auricular tachycardia exceeds a certain rate in this

a

b

c

d

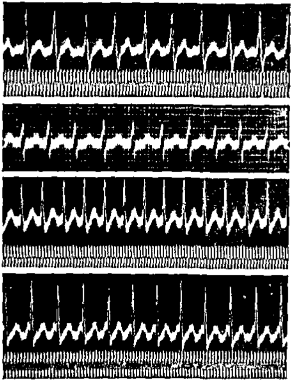


FIG 141a–d a and b show instances of paroxysmal auricular tachycardia c and d of auricular flutter

case the diastoles are very short and as was described in the chapter on sinus tachycardias the P waves of the extrasystoles may be hidden in the T waves of the preceding beats Then tracings occur like those of Fig 141a and b they illustrate the most common picture presented clinically in paroxysmal auricular tachycardia

If a tachycardia of this kind is recorded in the three leads then one cannot immediately decide from the electrocardiogram whether sinus tachycardia paroxysmal auricular tachycardia or flutter is present One must concede that Fig 130 (sinus tachycardia

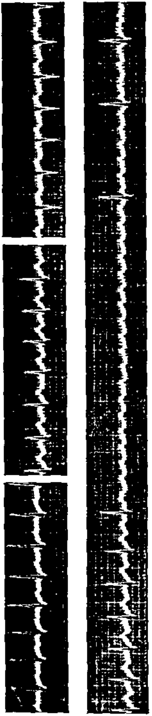


Fig 142 The upper tracing shows auricular flutter with 2 1 block in all leads The differentiation with certainty from auricular tachycardia was possible only by means of a carotid pressure test (lower tracing)

produced by the inhalation of amyl nitrite) Fig 139b (definitely paroxysmal auricular tachycardia) and Fig 141c and d (auricular flutter with 2 1 block) may resemble each other so closely as to be confusing In all three types of tracings we see slender, normally conducted ventricular complexes in which a P wave or a flutter wave may but need not be concealed

In very rare cases atrioventricular tachycardia (from the middle of the node) or ventricular tachycardia (above the bifurcation of the bundle of His) may show the same picture

Sinus tachycardia is excluded with relative ease This is immediately possible when the sudden beginning or the abrupt end of an attack is recorded or observed this never happens in sinus tachycardias If the patient presents a continuous fully developed tachycardia, a test should be made to determine whether an alteration of position of the body or slight exertion (repeated sitting up and lying down in bed) causes a slight acceleration of the pulse to appear which, as stated earlier, never fails in sinus tachycardia In paroxysmal auricular tachycardia or in auricular flutter, change of position of the body or slight exertion does not influence the rate In other words it is apparent that the diagnosis cannot always be made from the electrocardiogram alone, rather a functional test is necessary for this purpose

If no acceleration appears after slight effort, then auricular flutter with 2 1 block or paroxysmal auricular tachycardia is present Occa

sionally one may produce a doubling of the cardiac rate through increased effort and thus prove the presence of flutter with certainty (p 229) In other cases pressure on the carotid sinus provides evidence for differentiation In sinus tachycardia this pressure is often without effect or produces only a transient slowing which is maintained only for the duration of pressure Paroxysmal auricular tachycardia may cease during carotid pressure but *slight and transient slowing does not occur* (p 265) In auricular flutter the flutter waves may become visible through the disturbance of auriculo ventricular conduction since during the exertion of pressure the ventricular complexes which concealed the F waves may disappear

In Fig 142 a regular tachycardia is reproduced at first glance one cannot say whether it is an instance of sinus tachycardia paroxysmal auricular tachycardia or auricular flutter Sinus tachycardia could easily be excluded since the rate was unchanged after movement Auriculo ventricular conduction was depressed by means of pressure on the left carotid sinus with the result that distinct flutter waves appeared (lower curve)

Fig 143 Auricular tachycardia with variform P waves (frequently a preliminary phase of fibrillation)

But there are cases in which carotid pressure *and* exercise do not change the tracing then a distinction between auricular tachycardia and flutter cannot be made We shall see later that the therapy for these two conditions is identical so that an inability to distinguish them is not of therapeutic importance

Just as in ventricular tachycardias different forms of QRS complexes sometimes appear a peculiar type of auricular tachycardia exists in which the shape of the P waves varies At the same time an arrhythmia is also present Thus we see in Fig 143 constant variation of the diastole and changing form of the P waves Moreover, the P R interval changes continually and certainly in

connection with the length of the preceding diastole Sooner or later these tachycardias often are transformed into auricular fibrillation, just as the corresponding ventricular tachycardias (Fig 137) may pass into ventricular fibrillation.

In Fig 144 short sections from the electrocardiograms of two cases which were studied only during the tachycardia are reproduced As long as one is without knowledge of the electrocardiogram outside of the attack, the diagnosis of the type of tachycardia present in these cases is impossible Auricular flutter or auricular tachycardia might be present in which the ventricular complexes are abnormal

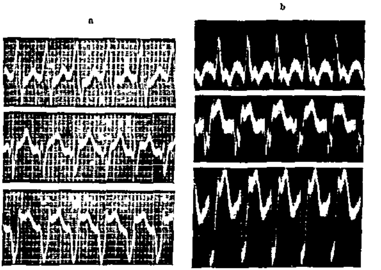


Fig 144a and b Two cases of paroxysmal tachycardia It is impossible to determine from the electrocardiogram shown what type is present

through simultaneous myocardial disease (organic disturbance of intraventricular conduction) The myocardium may be normal and the abnormality of the ventricular complexes might be merely the result of fatigue of certain branches of the conduction system ("functional disturbance of intraventricular conduction," aberration) On the other hand, ventricular tachycardia may be present In some cases one succeeds in recognizing a ventricular tachycardia by the venous pulse During ventricular tachycardia the auricles continue to beat normally in sinus rhythm In rare cases one recognizes the slow auricular rhythm by a mere inspection of the veins of the neck, but frequently the venous pulse curve must be recorded

Fig 145 is reproduced from a case of paroxysmal auricular tachycardia or flutter (?) in which entirely abnormal ventricular complexes temporarily appear through fatigue of a part of the intraventricular conduction system (the minute rate amounts to at least 300) with the result that one might assume a ventricular

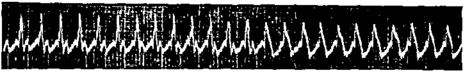


Fig 145 (Lead II) Paroxysmal auricular tachycardia (probably auricular flutter with 2 1 block) and in the second part of the tracing disturbance of intraventricular conduction

tachycardia was present The spontaneous transition of the normal ventricular complexes into abnormal ones may be seen in the record Since the irritability of the muscle fibres continually changes intraventricular conduction in such cases is at times normal at times abnormal All the time the tachycardia due to the abnormal stimulus formation in the auricle proceeds entirely without disturbance

Treatment of Sinus Tachycardia

It has been emphasized elsewhere that sinus tachycardia is not always a harmless phenomenon As a matter of fact at times it attains a very fast rate and for this reason must result in the same changes as other tachycardias with similar rates It also offers the disadvantage that on exertion an additional very unpleasant acceleration appears

No special treatment for sinus tachycardia is known Digitalis and quinidine which are widely employed and have recurrently been recommended are rarely useful No slowing appears even after the largest doses of digitalis and the patient experiences only the unpleasant effects of the drug Similarly quinidine treatment as a rule is ineffective The sole rational and effective treatment of sinus tachycardia is ætiological therapy, the treatment of the basic malady Sinus tachycardia after infections or intoxications vanishes when the basic malady disappears In hyperthyroidism a result can be secured by treating the thyroid hyperfunction but not from treatment of the heart itself A sinus tachycardia in hyperthyroidism which is unaffected by all treatment may return to normal cardiac action in a few days after the administration of iodine (as pre operative therapy) In cardiac neuroses the attention of the

physician is directed to the treatment of the entire individual and not to the heart alone

Also in cases of mitral stenosis with congestion but without auricular fibrillation in which extreme tachycardias may be precipitated by reflexes from the auricles in myocardial injuries and in coronary sclerosis with sinus tachycardia the slowing effect of digitalis on the heart is frequently not obtained Despite continuous digitalis therapy the rate does not fall below 100–120 These are the cases in which the appearance of auricular fibrillation brings relief because a slow ventricular action can then be obtained by means of digitalis

Treatment of Paroxysmal Flutter and Fibrillation

The clinical aspects of flutter and fibrillation (as continuous states) have already been discussed in detail The treatment of paroxysmal attacks of short duration will now receive consideration If they are frequent and appear almost daily quinidine may be given prophylactically and an endeavour made to find out the smallest effective amount of the drug Only when quinidine is not tolerated is digitalis administered usually the latter has a less certain effect But if the attacks as usually happens do not occur daily but only at longer and very irregular intervals a prophylactic regimen is of dubious value In these circumstances it is advisable to treat the attack when it has actually developed A dose of 0 25 gm (4 grs) of quinidine is given every two hours until the attack stops In most cases only 2 3 capsules are necessary Here also recourse must be had to digitalis when there is hypersensitivity to quinidine An intravenous injection of strophanthin (0 00025 gm 1/250 gr) may act very well In a majority of cases it abolishes the attack within thirty minutes

The appearance of *continuous fibrillation* which sooner or later replaces the paroxysmal attacks is very welcome to most patients since in these circumstances the patient no longer has constant anxiety about the attacks and success in dealing with the ventricular tachycardia is achieved with relatively small doses of digitalis

Clinical Aspects of Paroxysmal Auricular and Ventricular Tachycardia

Paroxysmal auricular and ventricular tachycardias are composed of auricular and ventricular extrasystoles For this reason in discussing the clinical aspects and therapy many points should be

repeated which have been mentioned earlier in the chapter on extrasystoles

The tachycardias vary considerably in their manner of appear ance and duration They occur at all ages Often the tachycardias have been compared to epilepsy In both states an attack may occur once in a life time, but it is also possible for them to recur daily Epilepsy as well as tachycardia may be the result of an organic disease, but both states may also be "idiopathic," that is, they appear without any ascertainable cause

In the tachycardias first one should always attempt to deter mine whether an auricular or ventricular tachycardia exists The auricular tachycardias are much more common than the ventricular, they also have a better prognosis If in a case of auricular tachy cardia the underlying pathological condition progresses, auricular fibrillation may develop this is not dangerous and represents a complication which is susceptible to treatment But if ventricular tachycardia is present, the appearance of fatal ventricular fibrillation is possible

In the ventricular tachycardias the ventricles contract alone *without preceding auricular action* Earlier (p 220) *reference was* made to the importance of the contraction of the auricles, par *ticularly in tachycardias for rapidly augmenting the ventricular* filling during diastole In other words ventricular tachycardias *are to be regarded less favourably than auricular tachycardias of the* same rate

There are two different types of auricular tachycardias At times (with a low rate) the auricles may contract so early in diastole, that they succeed in forcing some blood into the ventricles (Fig 138) But with increasing rates the P waves merge more and more with the T waves, that is, the auricles contract at a time when ventricular systole is still incomplete If the auricles contract when the ventricles are still in systolic contraction, the former cannot empty their contents into the ventricles Since the orifices of the great veins in the auricles are not protected by valves but are closed during auricular systole merely by an imperfect muscular mechanism, this obstruction is easily overcome, this readily leads to a reflux of blood into the veins The blood which has just returned from the veins into the heart is again ejected from the auricle back into the veins so that the liver rapidly enlarges and venous stasis in the neck becomes extreme When the auricular systole is 'engrafted' upon the ventricular it is called "superimposition" (*Wenckebach*) It is understandable, therefore, that the prognosis in tachycardias in

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which superimposition occurs is much more unfavourable than when it is absent Fig 130 shows clearly that above a certain rate, the diastole becomes so short that this superimposition must occur The rate at which this takes place is approximately 180, and this was called by Wenckebach the *critical rate* In tachycardias with lower rates superimposition *may* exist if, for example, conduction is prolonged In auricular tachycardias with a rate of 180 or more, it *always* exists

In a tachycardia at first the type (auricular or ventricular tachycardia) and then the rate must be investigated, in auricular tachycardia the presence of auricular superimposition must be determined, all these features are important

It is also essential to determine the frequency and duration of the attacks In one patient they recur at intervals of years, in another almost daily in some only during or before the menstrual period or in pregnancy (as extrasystoles), or only with marked constipation, meteorism, excitement, in others without any definite reason In one individual they last for minutes, in another for days, even for weeks Each case exhibits different behaviour The appraisal is easy when the patient comes with a long history and can furnish information in respect to all details Under these circumstances by careful inquiry one may gain an idea of the frequency of the attacks and their duration in the future The situation is different however when the patient comes *during or after the first attack* In this instance the prognosis should be determined only with great care Usually one will prefer to defer the decision until a period of observation permits sound judgment

If the patient reports that attacks have existed for years and the examination reveals normal cardiac findings, then one will, as in extrasystoles, judge the tachycardia as a harmless but annoying "functional" disturbance But if the patient is seen during or shortly after the first attack he must be observed for a time despite the negative outcome of the first examination, the tachycardia may be the first and only sign of an organic myocardial disease

Thus paroxysmal ventricular tachycardia, earlier than any other sign, may indicate coronary sclerosis a chronic myocarditis, or a diphtheritic myocardial lesion Since cases of coronary thrombosis have become more common, the ventricular tachycardias are a more frequent undesirable complication If one finds ventricular tachycardias with varying forms of the ventricular complexes, then, as mentioned earlier, an organic disease must always be suspected A digitalis effect must also be considered However, auricular

tachycardias with P waves of varying forms are always the result of an organic disease and never the effect of digitalis Usually they become transformed into fibrillation

Most patients who suffer from paroxysmal tachycardia perceive the sudden lightning like beginning and the abrupt end of the attacks so *distinctly that this form can by inquiry be differentiated from* other types of palpitation To be sure there are also patients who give vague reports and despite the existence of a tachycardia do not mention palpitation but only its resultant manifestations Premonitory signs which anticipate the beginning of the attack are rare

Anginal pain is a very common accompaniment of paroxysmal tachycardia *In the discussion of the consequences of tachycardia* in fibrillation it was stated that a diminution of the minute volume *to less than one half and a fall of blood pressure occur with rapid* tachycardia Since with increased rate of contraction the heart muscle needs a great deal more oxygen such increase of rate also leads to a *relative* diminution of cardiac blood supply when compared to cardiac performance This reduction of perfusion provokes pain which may show all transitions between slight burning and pressure to most severe angina pectoris (p 138)

Confusion of the anginal pain of coronary thrombosis with the anginal pain of paroxysmal tachycardia is not rare Severe pain maintained for hours in the midst of complete well being and without visible cause *appears in both states* (p 139)

The reduction of the stroke volume and the marked lowering of blood pressure appearing at the onset of the attack provide the reason for vertigo and states of weakness The attacks of unconsciousness are considered in the section devoted to Adams Stokes attacks (p 300)

The stasis which rapidly occurs behind the heart (in the *veins of the neck) and the resultant acute engorgement of the liver* may result in *vomiting* many patients complain especially about this symptom and other symptoms may remain in the background

A peculiar symptom at present scarcely investigated which accompanies paroxysmal tachycardia is *urina spastica* The patient reports voiding large amounts of clear watery urine This flood of urine often comes soon after the beginning at times during the *attack and rarely after its cessation The presence of this symptom* supports the diagnosis although it must be conceded that urina spastica occurs in other states and in disturbances of the vegetative nervous system

If the auricle contracts simultaneously with the ventricle as must happen especially in ventricular tachycardias superimposed waves appear in the neck veins, they may make themselves unpleasantly obvious to the patient as throbbing'

Fig 146 Ventricular extrasystoles in paroxysms

Extrasystoles in Paroxysms

In the forms of paroxysmal tachycardia discussed up to the present there may exist an interval of variable duration between the individual attacks at times this period of normal cardiac rhythm or at most isolated extrasystoles may persist for years But there is a variety of paroxysmal tachycardia which requires special mention It concerns cases in which the individual attacks are relatively short and last for only a few seconds or minutes One or two normal beats occur and then another attack of tachycardia begins This phenomenon is constantly repeated There may be scarcely two rarely several normal beats in sequence without extrasystoles appearing between them One short attack follows another so that these attacks were designated by Gallavardin as *extra systolie a paroxysmes tachycardiques*

Fig 146 shows these extrasystoles in paroxysms After two normal beats a series of four ventricular extrasystoles follows After two more normal beats a longer series of extrasystoles occurs

In other cases auricular extrasystoles with similar characteristics occur

In these patients the abnormal mechanism gives rise to multiple extrasystoles so constantly and stubbornly that even the first or second normal beat after the end of the tachycardia precipitates a new attack Every normal impulse acts as a new incentive scarcely has its action on the extra systolic centre declined when it precipitates the same disturbance again It is like a clonus appearing after every stimulation of the triceps muscle

Patients with this variety of extrasystoles at first make a good impression since the individual attacks last for only a short time and long post-extrasystolic pauses which are

always found after them balance the disturbance But it soon becomes evident that these cases must be viewed less favourably than the ordinary tachycardias If with the latter we succeed in *removing the attacks by measures to be described later (p 264)* we may anticipate that freedom from the symptoms will persist until the next attack and as a matter of fact this may never occur In extrasystoles in paroxysms which are under consideration in this chapter the situation is different Here also the administration of *large* doses of quinidine or digitalis is successful since the extra systoles vanish and sinus rhythm returns But if the dose of the drug administered is reduced even by a little the tachycardia recurs Since large doses of digitalis or quinidine cannot without danger be given over a long time and since small doses are inadequate soon it is realized that all therapy is useless for any length of time and *that it suppresses the abnormal mechanism of the heart only very transiently* Medicinal therapy is therefore ultimately abandoned and the future course of the patient varies There are cases in which these tachycardias slowly subside often only after years The individual attacks become progressively shorter and proportionately more normal beats are inserted between them If this is not the case however the heart is gradually injured by the steady acceleration which is maintained for years The dilatation of the heart which appears sooner or later leads to relative mitral or tricuspid insufficiency extreme stasis develops and finally death occurs The pathologist finds healthy valves and no signs of organic muscle alteration

The Cause of Paroxysmal Tachycardia

According to the results of available investigations the *origin* of paroxysmal tachycardia is attributed in most cases to a very frequent formation of stimuli *in the specific fibres of a stimulus* forming centre that is to the same event as gives rise to extrasystoles *Likewise they always begin at a definite interval after a normal beat like extrasystoles they are coupled*

Paroxysmal tachycardias have also been ascribed repeatedly to a circus movement and until very recently this explanation has been supported in many modified forms, but for most cases it certainly has no validity Before and after the tachycardia repeatedly *single extrasystoles are seen which show all the charac* teristics of those which compose the tachycardia Tracings which resemble Fig 186 cannot be explained by circus movement, since *the long pauses between extrasystoles are impossible if they are*

caused by a circulating wave such a wave must move incessantly In animal experiments it is easy to produce ventricular tachycardias and to prove that they originate in a circumscribed centre For example warming of this centre with a thermode accelerates the rate

The alternating tachycardias (see Fig 137) were frequently explained by an alternating action of two centres Recent investigations favour the existence of the formation of stimuli in *one* centre combined with disturbances of intraventricular conduction (Scherf and Kisch)

All transitions exist between single extrasystoles and paroxysmal tachycardia The difference is a matter of quantity and not of quality

The Treatment of Paroxysmal Tachycardia

The management of cases of paroxysmal tachycardia may be divided into the treatment of the attack itself and measures for the prevention of new attacks

If the patient is seen in an attack it is well not to have recourse to drugs at once but an attempt should be made to end the paroxysm by means of one of the following vagal reflexes

At first *carotid pressure* is tried Formerly it was called vagus pressure since the notion prevailed that the heart and the tachycardia could be depressed by pressure directly upon the cervical vagus and through direct excitation of the vagus But the experience of investigators has shown that pressure crushing or tearing of the vagal trunk exposed by an operation in the neck was entirely devoid of influence on the heart (Winterberg) Several cases have been reported in which light pressure on the neck certainly insufficient to influence the vagus trunk which lies deep behind the vessels of the neck depressed the heart and it has been shown that pressure was also effective in patients in whom the vagus had been previously sectioned by operation upon the side receiving the pressure (Scherf) Finally Hering proved in 1923 that no direct excitation came under consideration in so called vagus pressure but rather a reflex which emerged from the site of bifurcation of the carotid artery

Below the division of the common carotid into the external and internal carotid arteries there is a distinct expansion of the vessel the carotid sinus At this place the sinus nerve arises it unites with the glossopharyngeal and runs centrally with it to the vagus centre The two vagi represent the centrifugal path Mechanical stimuli and pressure on the carotid sinus lead to depression of the

heart and in a majority of instances cause attacks of paroxysmal tachycardia to vanish The pressure is applied in such a manner that it is exerted at the upper border of the thyroid cartilage upon the carotid artery (anterior to the M sternocleidomastoideus), which is pressed toward the vertebra Since immediate cardiac standstill may follow pressure, it is applied only with the patient in the recumbent position Moreover it is advisable to check the cardiac rate at the same time The degree of pressure must be adapted to the individual case In some patients very light contact with the skin suffices, in others quite strong pressure is necessary If the first endeavour fails, one may with advantage press somewhat higher or lower, because the exact site of bifurcation must be compressed pressure at any other place being ineffective Owing to anatomical variations, the site of bifurcation of the carotid artery is higher or lower in different people

In Fig 147a *there is at first* a regular sinus rhythm with a conduction time lengthened to 0 28 second The T wave of the seventh normal beat contains an auricular extrasystole which initiates a paroxysmal auricular tachycardia Fig 147b *shows* how the tachycardia is abolished *by carotid* pressure (right-sided) As long as merely light pressure was exerted the tachycardia con

a b

Fig 147a and b (a) shows the beginning of a paroxysmal auricular tachycardia In (b) the end of an attack (by carotid pressure) is shown

tinued but it stopped as soon as the pressure was increased Since the pressure on the carotid sinus was continued for some time the heart ceased to beat for a moment after the cessation of the tachycardia Two auriculo ventricular beats (without P waves see p 310) appear and finally normal sinus rhythm returned After the long pause the conduction time is shortened owing to better recovery of the conduction system The cessation of carotid pressure is indicated on the white line above the electrocardiogram (stimulus signal)

Pressure is usually effective on the right side But there are cases in which it also succeeds or succeeds *only* upon the left side If the rules given are observed carotid pressure is devoid of danger

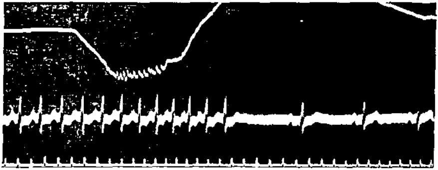

FIG 148 Termination by deep inspiration of an attack of paroxysmal auricular tachycardia

It is obvious that pressure must not be exerted upon both sides simultaneously

Many patients soon learn to exert the pressure themselves and can immediately abolish the attacks without medical assistance

If carotid pressure is ineffective a trial is made with another vagal reflex There are patients who can abolish an attack by deep inspiration or by holding the breath others succeed through the Valsalva experiment (the patient is advised to press down as in defæcation) Bulbar pressure is very effective especially in young people The patient looks downward closes the eyes and then pressure is exerted with increasing force on both eyeballs

In Fig 148 there exists a paroxysmal auricular tachycardia which could be confused very easily with auricular flutter with full conduction Each attack was ended by deep inspiration (inspiration is shown in the respiratory curve with the excursion upward)

Finally there are patients who can immediately end a paroxysm by inserting a finger into the throat in order to produce vomiting

Often they learn this without instruction In the course of their attacks acute hepatic stasis may occur which produces a sense of nausea and vomiting The patients note once or twice that the attack ends with the initial retching Subsequently they no longer wait until the vomiting comes spontaneously, but attempt to produce it immediately by inserting the finger into the throat, thus causing an emetic reflex

Repeatedly one encounters some patients in whom one reflex, and other patients in whom a different reflex is more effective in a few minutes all are tested Only when they fail is *medicinal therapy* necessary Here quinidine should be mentioned in the first place, it can be injected during the attack or may be given by mouth

An injection of quinidine may have an almost magical effect (Hecht and Zweig Singer and Winterberg) The result appears so rapidly that at times the attack ceases before the injection is completed Nevertheless great caution is necessary since most profound collapse may be observed after the intravenous injection of quinidine

Physicians who dwell in malarial regions and who inject large amounts of quinine are aware that now and then a death occurs after *many injections were well tolerated* If for example 0 2 gm (3 grs) of quinine or quinidine is given to a healthy dog only very *slight effects are seen* But the same amount in another animal, with the experiment continued for a longer time with loss of blood *or a damaged heart, may result in diastolic cardiac standstill* If a patient seeks relief in an attack and the status of his heart outside of *the paroxysm is unknown, an injurious effect of the injection is* quite possible Since in an attack one simply hears a pendulum *rhythm of the sounds which rapidly follow one another and one* cannot hear murmurs or other abnormal phenomena of auscultation *a profound alteration of the myocardium or a marked valvular* lesion may thus not be recognized Owing to the small stroke *volume and the low blood pressure during the attack the second sound is often inaudible*

We should *also realize that myocardial injury may occur during* the attack and may be caused by it in otherwise healthy people (p 138) *If for some other reason quinine is injected it is advisable* to remember these possibilities The initial injection should not exceed 0 2 gm (3 grs), *this amount may be increased the next* time to 0 5 gm (7½ grs) at most These larger amounts are administered only *when the small dose was well tolerated Quinine must* be injected slowly

In recent years well tolerated quinine preparations for intra

muscular injection have made intravenous injections superfluous One can inject 0 5 gm at a time intramuscularly without danger and if it is necessary may repeat this amount once or twice on the same day To be sure such an immediate action cannot be expected as after an intravenous injection In most cases success is attained without recourse to injections, quinidine sulphate in amounts of 0 25 gm may be administered orally and this dose repeated every two hours until the attack is abolished, usually this occurs very soon

However if the patient does not tolerate quinidine by no means a rare event in the presence of hypersensitivity, then digitalis and strophanthin are better alternatives The employment of these drugs in some cases affords such great advantages that they may be used instead of quinidine even when it is not contra indicated

If a rapid action is urgently necessary it is advisable to administer an intravenous injection of ¼ mg (1/250 gr) of ouabain The result is obtained with unusual rapidity, at the latest in half an hour If the patient has not been previously treated with digitalis an injurious action need not be feared Even when the injection does not abolish the attack it may impair conduction and lead to periodic dropped beats or to a 2 1 block that is to a useful and considerable slowing of the rate of the ventricles

Carotid pressure which has previously been ineffective applied shortly after an injection of strophanthin may end a paroxysm This effect is comprehensible since the investigations of Hering and F Kisch have shown that the carotid sinus is sensitized by digitalis

In less urgent cases the oral administration of digitalis is recommended In this instance, to be sure, larger doses than are ordinarily employed in digitalis therapy are necessary as a rule It is advantageous to administer rapidly acting preparations (pure glucosides) in amounts which correspond to 0 4–0 6 gm (6–9 grs) a day of the assayed pulverized digitalis leaves If in the course of therapy of an auricular tachycardia a partial block between the auricle and ventricle develops often a marked reduction of the dose of digitalis succeeds in maintaining it Very often paroxysmal tachycardia vanishes completely during digitalis therapy In cases of *extrasystolie a paroxysmes tachycardiques* it is especially effective and as a rule superior to quinidine therapy

On the contrary quinidine is preferable to digitalis when a patient suffers from the paroxysms at regular intervals, because in this instance the attacks may be prevented by prophylactic treatment One proceeds exactly as in extrasystoles and endeavours to find the smallest amount sufficient to prevent the appearance of tachycardia

The combination of digitalis with physostigmine has often been recommended in order to reinforce that component of digitalis action which affects the vagus In this case physostigmine is administered orally in doses of 0 5–1 5 mg (1/120–1/40 gr) daily, this dose should not be exceeded, because of unpleasant untoward effects We are unable to perceive any definite advantages by its use

Apart from quinidine digitalis and strophanthin a large number of other remedies have been recommended for the treatment of paroxysmal tachycardia It must be conceded that many other substances which act upon the myocardial cells can also arrest tachycardia *This is true of atropine but in large doses it produces* unpleasant effects Acetyl β methylcholine may also be tried which is effective also after hypodermic injection unfortunately it has occasionally unpleasant untoward effects Even apomorphine has been recommended to stimulate the vomiting centre and in this way the vagus But those who have once observed the unpleasant mode of action of apomorphine will no longer utilize this agent *ipecac in emetic doses is more worthy of recommendation* (Weiss and Sprague) Vomiting is easily induced by sticking the finger into the throat it is a simpler procedure and equally effective The recommendation to treat tachycardias by adrenalin is incomprehensible perhaps it occasionally abolishes the tachycardia but more often it may precipitate fatal ventricular fibrillation

Magnesium sulphate is effective in auricular tachycardia and in this condition not dangerous (Zwillinger) Ten to twenty cubic centimetres of a 15 per cent solution are slowly injected This agent is still in the trial period and additional extensive experience must be awaited Magnesium sulphate is recommended particularly for tachycardias and extrasystoles following excessive digitalization As a matter of fact it does not act in every case and its effect is usually transient It has proven beneficial to us in paroxysmal auricular tachycardia Whether or not ventricular tachycardias will respond favourably to the remedy is still uncertain

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DISTURBANCES OF STIMULUS CONDUCTION

Introduction , Escaped Beats

The capacity for conducting stimuli is a fundamental property which *every* myocardial fibre possesses regardless of whether it belongs to the common muscle or the specific tissue Every stimulus originating in or applied to a place in the cardiac muscle spreads over the heart it is conducted as soon as it is above threshold

According to the prevailing conception, conduction in the heart is *myogenic* *In the excitation of a muscle fibre, electrical de* polarization appears on the limiting membranes The process of depolarization on one fibre acts as a stimulus for a neighbouring fibre etc In other words the excitation is transferred from one *fibre to another* *Since with the excitation of a healthy muscle* fibre a contraction is necessarily associated a contraction wave passes over the heart with the wave of excitation Actually with an appropriate experimental arrangement it was possible to demonstrate in the ox s heart that after the auricular and before the ventricular contraction a contraction of the bundle of His and its branches takes place (Ishihara and Nomura)

In normal hearts the original stimulus is formed in the sinus node from whence it spreads over both auricles through groups of muscle fibres that radiate in all directions By means of the A–V system the stimulus reaches the ventricles Since every area of the myocardium is capable of conduction a disturbance of this event may occur anywhere The results of this disturbance of conduction vary according to the site and size of the pathological focus Naturally such a focus in the wall of the auricles will have much less significance than an equally large focus in the A–V system

As a matter of fact disturbances of conduction are remarkably rare when one recalls how commonly myocardial diseases occur and how frequently one finds disturbances of stimulus formation (fibrillation extrasystoles) There are several reasons for this rarity A knowledge of the anatomy of the specific tissue assists in making this comprehensible

It was stressed earlier (p 8) that the sinus node is connected

with the auricle by so many fibres that even when many of the junctional fibres become affected, a sufficient number remain intact or functionally efficient, so that the normal sequence of the heart beat is not disturbed For this reason sino auricular disturbances of conduction are rare

As stated earlier (p 12), the A–V system for the greater part of its course is separated from the common muscle, moreover it possesses *a special blood supply which comes from the right and* left coronary arteries For this reason it has its own pathology Very often the ventricle is found thickly studded with scars or the cardiac muscle severely degenerated (pernicious anæmia) while the A–V system is entirely normal On the other hand, severe alterations are at times found exclusively in the specific system, in otherwise normal hearts if for example, only the arteries of the specific tissues are affected

Moreover, anatomical investigations show that the right and left main trunks of the A–V system are built in an entirely different manner, so that a small pathological focus which renders the right bundle branch completely incapable of conduction, scarcely injures the left Furthermore, the blood supply of the two bundle branches is entirely different The fact that the conduction system possesses excellent vascularization and that branches from the right and left coronary arteries anastomose with each other at many places, is undoubtedly the reason why disturbances of conduction appear so *seldom in spite of the great prevalence of myocardial diseases (which* are mostly ascribed to a vascular disorder)

Besides these anatomical factors the physiological properties of the heart muscle also play a part, and they together are responsible for the rare appearance of disturbances of conduction and, if they have occurred, for the lack of harmful results, as a matter of fact often their presence is not even noted

For a long time the opinion was advanced that conduction *in the heart is undisturbed and proceeds with normal velocity* only when a sufficiently wide path is available Narrowing of the diameter of the pathway in an area in the A–V system was supposed to lead to disturbances of conduction (Gaskell) Recent investigations performed with more modern methods, however, have shown that the width of the path does not determine the velocity of conduction and that as long as *one* fibre is intact in a cross section, the conduction remains entirely normal

Only when this last fibre is also affected does a disturbance of conduction appear Conduction in the myocardium is called

"auxomeric", this means that the stimulus can be transmitted from one fibre to any optional number (v Kries)

As a result most profound lesions of the bundle of His need not produce disturbances of conduction as long as a single fibre in the cross section is intact

At times complete heart block appears overnight Nevertheless, histological examination undertaken shortly after shows the presence of very old and advanced lesions Conduction was normal as long as one fibre of the cross section remained intact, as soon as this became involved conduction was interrupted

But even when a complete interruption of the bundle of His prevents the stimulus from reaching the ventricle, the latter does not stand still because the automatism of the deeper centres immediately becomes active Every specific fibre can form rhythmic stimuli as discussed earlier (p 165) stimuli normally are formed only in the head of the sinus node and all the other specific fibres remain inactive If for some reason an interruption of conduction occurs a centre situated below the site of the block immediately becomes active and assumes control as the most rapidly acting area of stimulus formation This automatism acting as a protective mechanism prevents standstill and thus prevents the patient from perceiving any unpleasant sensations despite heart block Only rarely does automatism fail (p 301)

In Fig 149 a marked respiratory arrhythmia may be seen The tracing was recorded in a child During expiration the activity of the sinus node was so slow that a stimulus from the A–V node became effective (p 311) During the slow rhythm beats occur which are not preceded by P-waves These contractions which serve to rescue the patient when stimuli from the higher centres fail to appear, are called *escaped beats* These escaped beats are not pathological but rather show that the automatism of the deeper centres is well developed

In Fig 150, after every two normal beats an auricular extra systole appears (it is recognized by the negative P wave coinciding with the T wave), it is not conducted to the ventricle, but is blocked In the following post extrasystolic pause an escaped beat emerges from the A–V node, to be sure, some hundredths of a second later the post-extrasystolic normal beat would have arrived, its P wave may be seen in the tracing between the QRS complex and the T wave of the escaped beat

Also during pressure on the carotid sinus, through which sinus node activity is depressed the deeper centres lying in the A–V node may become active and prevent cardiac standstill

Fig 151 shows the effect of carotid pressure on the heart The *electrocardiogram (Lead III) was obtained from a healthy person*

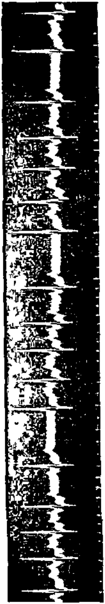

Fig 149 Respiratory arrhythmia with escaped beats during the bradycardia

Fig 150 After two normal beats a blocked auricular extrasystole appears the long post extrasystolic pause is ended by an escaped beat

Fig 151 Sinus node activity is depressed by carotid pressure and escaped beats appear

The white perpendicular line indicates the beginning of carotid pressure The activity of the sinus node is immediately inhibited and deeper automatic centres assume control in place of sinus stimuli thus preventing a prolonged ventricular standstill

Some very important forms of disturbance of conduction have already been considered. In the initial chapters the disturbances of intraventricular conduction were described, that is disturbances of conduction below the site of division of the bundle of His (bundle branch block, arborization block, wide initial deflections and abnormal T waves). As a rule these are not characterized by a disturbance of auriculo-ventricular beat sequence, since even a complete interruption of conduction in the specific tissue of one ventricle leaves the connection to the other open. Only in disease of *both trunks is the path from the auricle blocked*. However, this is rare.

The Various Forms of Disturbance of Atrioventricular Conduction

When a disturbance of conduction is mentioned, one generally understands in clinical parlance a disturbance of conduction in the

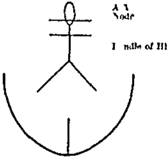

FIG 152 Schematic drawing of the conduction system

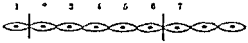

FIG 153 Explanation in text

bundle of His above the bifurcation or in the A-V node; this constitutes a disturbance of atrioventricular conduction whose various forms will be considered in the following discussion.

Let us assume a pathological focus develops in the A-V system, for example in the course of rheumatic myocarditis. With increasing damage to the conduction path the passage of stimuli becomes increasingly difficult and the mildest form of disturbed conduction gradually is transformed into more severe disturbances.

There exists an incomplete (partial) block as long as some stimuli still reach the ventricle from the auricle; the block is complete when auriculo-ventricular conduction is entirely interrupted.

To facilitate comprehension the events in auriculo-ventricular conduction are presented by means of two drawings. In Fig 152

the A–V system is indicated the other drawing (Fig 153) represents a chain of fibres of the A–V system Let us assume that the pathological focus lies between the two horizontal lines of Fig 152 and the perpendiculars of Fig 153 *Above and below, to the right and left of these limits*, the conduction system is normal

Since a disturbance of conduction appears only if the disease impairs every fibre of the cross section a drawing of the type of Fig 153 is a permissible indeed *according to the law of auxomeric conduction is the only possible way to explain* abnormal conduction events

As soon as the affection begins in the tissues demarcated by the lines the last healthy muscle fibre 1 in Fig 153 will excite the first altered fibre 2 in a normal manner, the pathological fibre however

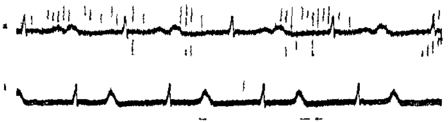

Fig 154a and b (Lead II) Two examples of a marked prolongation of conduction time In the tracing (b) the P wave is concealed in the preceding T wave

responds to the stimulus slowly and likewise the stimulus response in general will be prolonged in the remaining diseased tissue But as soon as the stimulus again reaches healthy tissue (fibre 7) conduction will again proceed as rapidly as normal Owing to the retardation in the diseased tissue the ventricle will be excited later than normal and the auriculo ventricular conduction time the P–Q or P–R interval is prolonged *This prolongation of conduction time* is the first and simplest grade of disturbed conduction This *retardation (prolongation of conduction delayed conduction)* may be only trifling so that conduction may require 0 22–0 24 second, but conduction times of 0 4–0 6 second and more are known But since *every* auricular stimulus even if slowly reaches the ventricle there is no disturbance of rate or rhythm

Examples of prolongation of conduction time are seen in Figs 134 and 147

In Fig 154a the conduction time is prolonged to 0 55 second, so that a P wave appears soon after the T wave If the conduction time is longer or the rate faster as in Fig 154b (P–R = 0 54 second),

the P wave is concealed in the T wave (rarely even in the initial deflection of the preceding beat)

If the pathological process progresses in the A V system and the specific fibres themselves are more severely injured it may happen that the first affected fibre (fibre 2 in the drawing) on occasions does not respond to the stimulus and for this reason a stimulus is blocked on its way to the ventricle accordingly a ventricular beat is dropped and the P wave is blocked Owing to the fact that the specific tissue did not conduct on one occasion it may recover to such an extent that the stimulus following the blocked P wave is conducted much better in fact often normally But owing to fatigue of the junctional fibres conduction time soon increases and after some time a block recurs This form of periodic dropped beat is called a Wenckebach period since Wenckebach (1899) discovered it

Fig 155 Wenckebach period

in an ingenious way by means of an analysis of the radial pulse alone A Wenckebach period is characterized by the following facts despite rhythmic auricular activity periodically a ventricular beat is dropped the conduction time before the dropped beat being prolonged and after the dropped beat being shortened (owing to recovery) Such a period may be long and many beats may reach the ventricle before one is blocked but it may also be so short that every sixth fifth or third beat may be blocked The length of a period may also vary continually in the same case The increase of conduction time may develop gradually or suddenly the conduction time may be greatly prolonged or may show only slight prolongation All possible variations occur

In Fig 155 a Wenckebach period is reproduced it appeared in a case of rheumatic myocarditis without digitalis therapy The periods are short and at times every third beat is dropped The auricles contract regularly

In Fig 156 a longer period may be seen where every fifth to ninth beat is blocked The conduction time increases slowly The

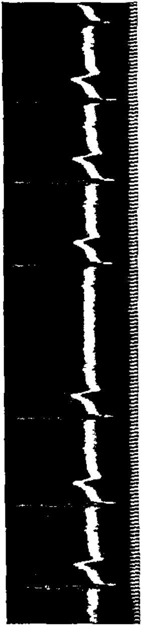

Fig 156 Wenckebach period

Fig 157 Paroxysmal auricular tachycardia with 2 1 block and a Wenckebach period One stimulus is abnormally conducted *within the ventricle*

P waves are splintered (disturbance of intra auricular conduction, p 295) *The tracing was obtained from a patient with a mitral valve* lesion who had been treated with massive doses of digitalis

In Fig 157 there is a paroxysmal auricular tachycardia with a disturbance of conduction from auricle to ventricle. In tachycardias, just as in auricular flutter, these disturbances of conduction are common The auricular rate amounts to 187. At the beginning of the tracing there is a 2 1 block ; then one beat is quickly, the next slowly and aberrantly conducted to the ventricle (a branch of the conduction system below the bifurcation of the bundle was not functioning), only the third stimulus is blocked Thereafter a 2 · 1 block occurs, and then another short period This disturbance

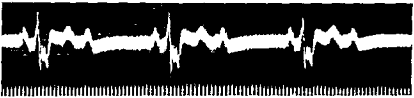

FIG 158 (Lead II) 2 . 1 block and disturbance of intraventricular conduction

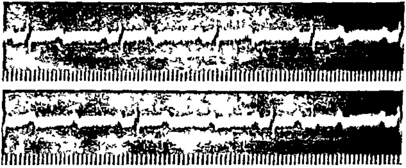

FIG 159a and b (Lead II) In Fig. 159a there is a 2 1 block which is transformed into a 3 1 block (in b) after the inhalation of amyl nitrite.

of conduction immediately vanished when the tachycardia reverted to sinus rhythm

In the shortest possible period every third beat is dropped The conduction of the second stimulus is prolonged in comparison to the first, and the third beat is blocked. If the disturbance increases then the bundle becomes so fatigued even after the passage of one stimulus that the next one is not conducted ; a 2 : 1 block ensues and every second stimulus remains blocked. In a 2 : 1 block the stimulus may be conducted with normal or delayed velocity. With further impairment of conduction, only every third or fourth stimulus is conducted (3 : 1, 4 : 1 block, etc).

Fig. 158 shows a 2 : 1 block. Moreover, there is a marked intra-

ventricular disturbance of conduction with widening and slurring of the initial deflections

In Fig 159a a 2:1 block also exists At first glance it looks like a normal electrocardiogram It is suspicious however, in that the form of the T waves resembles that of the P waves and that the *P–T intervals are the same as the T–P intervals* Since this may happen normally it cannot be employed as proof of a 2:1 block In order to establish the diagnosis, another tracing must be recorded after exercise or after inhalation of amyl nitrite

Fig 159b shows that auricular activity increased a little after the inhalation of amyl nitrite thereby the demands upon the conduction system were *increased which thus became more easily fatigued* so that a 3:1 block appeared If normal sinus rhythm had been present only a simple acceleration of rate would have occurred after amyl nitrite

A 2:1 block also is present in Fig 160 On first inspection one gains the impression of blocked auricular extrasystoles The P–P interval which includes a ventricular beat is approximately 0.72 second long while those which appear subse*quently are about 0.88 second The blocked* P wave comes soon after the T wave and is premature But the same disturbances of auricular rhythm are occasionally found in 2:1 block or with complete block, since the interval between two auricular waves which includes a ventricular systole is often shorter than one which does not No satisfactory explanation for this phenomenon has yet been put forward (Kauf) A vagal reflex *seems to exist since the systolic increase of blood* pressure brought about by the aortic and carotid nerves leads to an increased vagal tone which becomes manifest only during the following cycle

Fig 160 (Lead II) 2:1 block with auricular arrythmia

Fig 161 shows a regular 4:1 block in Lead III

Much higher grades of block are also possible for example there may be 10:1 block in which only one of ten stimuli is conducted from the auricle to the ventricle after which the bundle recovers so slowly that the following nine beats are blocked In

high grade block the ventricles naturally do not remain inactive until the fifth or tenth stimulus is conducted. In marked heart block rarely in 3:1 but usually in 4:1 the deeper centres become active during the longer ventricular cycles. A centre lying below the area of block develops its own automatism and may, since it is no longer depressed by more frequently conducted stimuli, assume control of the ventricle.

In Fig 162 a 7:1 and then 2:1 block is seen. Every seventh and later every second auricular stimulus reaches the ventricle with a conduction time prolonged to 0.30 second. The depressed S-T interval and the inverted T wave of the conducted beats (in Lead II) indicate myocardial damage. In the long pauses created by the block a more deeply situated automatic centre begins to function. Since the stimuli formed by this abnormally situated centre spread abnormally within the ventricle the related ventricular complexes appear altered.

If the conduction to the ventricle fails entirely, the auricles beat

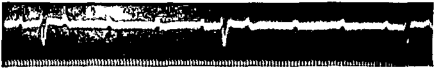

Fig 161 4:1 block

as under normal conditions in sinus rhythm but the ventricles contract with complete independence under the control of their own automatic centre. In this instance there is a complete block or a *total dissociation of the two rhythms*.

In an electrocardiogram complete block is recognized by the regular auricular and regular ventricular activity but *continual variation of the P–R interval*. As will be shown later the rate of the ventricle may vary.

In Fig 163 the auricles and ventricles contract with perfect regularity. The auriculo-ventricular (P–R) intervals vary continually so that it must be assumed that the ventricles are working independently of the auricles and with their own rhythm. In other words a complete auriculo-ventricular block is present. The ventricular rate amounts to approximately 50 per minute.

In Fig 164 a regularly contracting auricle and a regular ventricular contraction with varying auriculo-ventricular intervals may also be seen. The ventricles contract very slowly, their rate being 18 per minute.

Fig 162 7 1 block then 2 1 block In the long ventricular pauses a deep ventricular centre becomes active

Fig 163 Complete auriculo ventricular block

Fig 164 Complete auriculo ventricular block

The abnormal appearance of the ventricular complexes in some cases of heart block does not indicate myocardial injury It is due to the fact that owing to the heart block deep centres assume control over the ventricles and in view of their abnormal location the excitation spreads abnormally within the ventricles Only when the automatic stimuli originate above the bifurcation of the bundle are the QRS complexes normal

Another form of auriculo ventricular block will be considered later (p 290)

Clinical Aspects of Disturbances of Atrioventricular Conduction

The ætiology of auriculo ventricular conduction disturbances may vary considerably It is convenient to differentiate between three groups —

1 There are disturbances of conduction in a perfectly healthy heart They are called—not very aptly—functional disturbances of conduction and are found in conjunction with various disturbances of rhythm and are essentially dependent upon two factors

They appear (*a*) when demands are made upon the conduction path very early in diastole this for example occurs not rarely in extrasystoles Reference has previously been made to the fact that auricular extrasystoles may be blocked or may be conducted aberrantly if they are very premature These disturbances of conduction are not an indication of disease they occur in perfectly healthy hearts since the healthy heart and healthy conduction tissues have a refractory period and for this reason require a certain amount of time for recovery If the atrioventricular conduction system is stimulated prematurely it either conducts poorly or not at all

Conduction disturbances also appear (*b*) when too many stimuli per minute are presented for conduction

The functional capacity of a conduction pathway permits response to demands only up to a certain limit It can be shown experimentally that when a demand is made upon the bundle of His to conduct only 100 beats per minute the conduction time increases a little (Lewis and Master) But if higher rates occur as in a tachycardia or if auricular flutter with an auricular rate of 300 or auricular fibrillation with 600 per minute develops the conduction path between the auricle and ventricle is fatigued by overwork Naturally it is very advantageous for the patient when the ventricle does not respond with a high rate If in auricular fibrillation all the auricular stimuli reached the ventricle death would follow immediately In auricular fibrillation an incomplete auriculo ventricular block

always exists, as soon as the fibrillation or tachycardia ceases auriculo ventricular conduction again becomes normal These functional disturbances in extrasystoles tachycardias flutter and fibrillation of the auricles are described in their respective sections (pp 221 and 229)

In other words it is evident that a definite period of recovery *is necessary for normal conduction* The more profoundly conduction is impaired, the longer this recovery time must be For this reason it is comprehensible that disturbances of conduction appear much later in bradycardias than in tachycardias In a combination of an auricular tachycardia and injury to the A–V system disturbances of conduction appear very regularly and early Disturbances of conduction may vanish when the heart rate slows

Besides these disturbances of conduction in which the pathway is normal there exist two other groups in which the conduction path *is actually damaged*

2 There are disturbances of conduction from the effect of poisons on the A–V system As an example digitalis deserves particular emphasis Other poisons scarcely come under consideration in the clinic For a long time it was believed that salicylates could produce disturbances of conduction but more recent investigations have shown that even when salicylates are given in large amounts they do not influence conduction in the heart (Master)

Digitalis impairs conduction in two ways It acts upon the muscle itself reducing the irritability of the fibres and prolonging their refractory phase Moreover, it depresses conduction through an increase of vagal tone (p 223) All degrees of incomplete and complete block can develop from the effect of digitalis

The status of the heart muscle is of importance in regard to the appearance of digitalis extrasystoles, this is equally true of disturbances of conduction While very large amounts of digitalis are necessary to induce disturbances of conduction during *health in a cardiac patient at times they occur even after small doses There* are patients who show a disturbance of conduction even after three powders of 0 1 gm of digitalis

If digitalis therapy is continued in spite of the appearance of a disturbance of conduction there are two possibilities Either the disturbances increase and a higher degree of block appears or the disturbance vanishes and gives way to normal rhythm Since the status of the heart muscle plays an essential role in the occurrence of digitalis conduction disturbances, it is possible that the improvement

accompanying a progressive digitalization removes the sensitivity of the conduction tissue to digitalis and even the existing conduction disturbance may vanish In other words a behaviour similar to that already discussed in digitalis extrasystoles is also encountered in this connection

If disturbances of conduction appear during digitalis therapy they do not present a contraindication to the continuation of therapy An incomplete block may be increased or transformed into a complete block But this does not constitute a danger since even with high grade block and with complete interruption of conduction between the auricles and ventricles, a normal circulation may exist

Moreover it is incorrect to withhold digitalis from a patient needing it simply because a disturbance of conduction from other causes is present If for any reason digitalis is indicated, it should be given despite disturbances of conduction Indeed, in patients with auricular fibrillation an endeavour is regularly being made to produce an increase of the conduction disturbance by means of digitalis those cases of auricular fibrillation which exhibit a severe disturbance of conduction after small doses of digitalis for this reason often respond particularly well to digitalis therapy It would be incomprehensible if the production of conduction disturbances were aimed at in auricular fibrillation, but were feared as a complication in sinus rhythm

Digitalis can produce all forms of incomplete and even complete heart block For this reason in every patient with a disturbance of conduction an inquiry should be made concerning previous digitalis therapy The disturbance may persist for several days after the cessation of treatment

3 One last form of disturbance of conduction depends upon an organic disease of the A V system It is not a very rare event to find a congenital heart block in patients with a congenital defect of the ventricular septum The bundle of His lies in the immediate neighbourhood of the septum membranaceum In the absence of the connective tissue septum and its adjacent muscles the anlage of the bundle of His may be disturbed

A structural injury of the conduction system may appear in the course of a toxic or degenerative diffuse damage of the heart muscle, it may be caused by foci located in the conduction system and finally may be the result of a disease of the vessels which nourish the specific tissues All kinds of pathological processes in the myocardium may also appear in the specific tissues, among others, diphtheria rheumatic myocarditis, gumma, tuberculosis myo

malacia, carcinomatous metastasis may be present In children and young people, usually diphtheria or rheumatic disease may be blamed, in older individuals coronary sclerosis is most often responsible It should be stressed once again that a disease of the common muscle need not always involve the specific tissue and on the other hand, the specific tissue may be severely affected although the working muscle is normal These events are explained by the isolation of the conduction tissue from the common muscle and by the fact that the specific tissue has its special blood supply

When the history reveals the absence of previous digitalis therapy, *a careful investigation must be undertaken to determine* which *organic* cause is responsible for the disturbance of conduction

The *clinical diagnosis* of many forms of disturbances of conduction is not possible without the employment of graphic methods In a simple prolongation of auriculo ventricular conduction there is no alteration of the rate or rhythm to make the diagnosis possible But not rarely attention is drawn to the long conduction time through the appearance of gallop rhythm The auricles also produce sounds which are normally inaudible, since they merge with the first heart sound In prolongation of conduction time the auricular sound occurs so early in diastole that it becomes audible as a third heart sound after the two normal sounds This gallop rhythm vanishes *immediately when conduction again becomes normal*

Wenckebach's periods can on clinical examination at least be suspected under the following conditions when a patient with rhythmic cardiac action receiving digitalis or an individual shortly after having had diphtheria or a myocardial disease suddenly manifests periodic omissions of ventricular systoles in contrast to extrasystoles the pauses caused by dropped beats not being preceded by *premature contractions*

In most cases the diagnosis of a complete heart block also is possible without an electrocardiogram In the first place it is *important to know that in heart block a bradycardia need not* always exist In former years heart block was considered only when a decided *bradycardia* was present, and it was assumed that every case of heart block manifested bradycardia. But it has been pointed out that in complete block a very *fast ventricular rate* may be present and, for reasons mentioned later, this is very commonly the case In other words bradycardia is not the chief sign of complete block

Even when a marked bradycardia exists heart block need not be present, numerous other conditions may occur, and knowledge of them is important

accompanying a progressive digitalization removes the sensitivity of the conduction tissue to digitalis and even the existing conduction disturbance may vanish In other words a behaviour similar to that already discussed in digitalis extrasystoles is also encountered in this connection

If disturbances of conduction appear during digitalis therapy they do not present a contraindication to the continuation of therapy An incomplete block may be increased or transformed into a complete block But this does not constitute a danger since even with high grade block and with complete interruption of conduction between the auricles and ventricles a normal circulation may exist

Moreover it is incorrect to withhold digitalis from a patient needing it simply because a disturbance of conduction from other causes is present If for any reason digitalis is indicated it should be given despite disturbances of conduction Indeed in patients with auricular fibrillation an endeavour is regularly being made to produce an increase of the conduction disturbance by means of digitalis those cases of auricular fibrillation which exhibit a severe disturbance of conduction after small doses of digitalis for this reason often respond particularly well to digitalis therapy It would be incomprehensible if the production of conduction disturbances were aimed at in auricular fibrillation but were feared as a complication in sinus rhythm

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Even when a marked bradycardia exists heart block need not be present numerous other conditions may occur and knowledge of *them is important*

A very common form of bradycardia is the constitutional or hereditary bradycardia which is found in many families In such cases the cardiac rate may be below 50

Usually a bradycardia develops in athletes during training An increase of cardiac performance can be obtained through an increase of the stroke volume (increased flow to the heart) or through an increase of cardiac rate (within certain limits) Since the oxygen consumption of the heart increases disproportionately when the second possibility is involved the first is more rational Actually this occurs in athletes whose circulation is trained A bradycardia with a correspondingly larger stroke volume belongs to the *athletic heart*

There are also toxic bradycardias The bradycardias of jaundice and of hunger œdema are well known In some influenza epidemics a bradycardia prevails and disappears only after weeks (in other epidemics of grippe there is a toxic sinus tachycardia)

Bradycardia can also be produced by the vagus It may develop from increased intracranial pressure (tumour meningitis) which acts directly upon the vagus centre but a reflex from the carotid sinus (glands in the neck neck tumours) may also be responsible The vagal bradycardia is characterized by a slight sinus arrhythmia a feature to which Wenckebach called attention it may be extreme in some cases (brain tumours)

Regardless of whether or not a bradycardia exists a *heart block* can by means of several signs be recognized without an electro cardiogram In the first place the observation of the venous pulse with simultaneous study of the cardiac rate may reveal venous undulations at the time of ventricular diastole This is explained in the following manner the auricles which contract more frequently than the ventricles produce an auricular wave the *a* wave To be sure this phenomenon is not characteristic of *complete* block for it is also present in 3 1 4 1 block etc

Another sign furnished by the venous pulse is much more characteristic In complete heart block the auricles and ventricles work independently of each other each with its own rhythm This results in *interference* whereby an auricular systole periodically occurs at the same time as the ventricular systole so that in an electrocardiogram a P wave is concealed in a QRS complex or a T wave But if the auricle contracts when the ventricle is still in systole it results as was described earlier (p 259) in superimposition which produces a large venous wave in the neck If with regular ventricular action an irregular venous pulse occurs and (when the blood is forced from the auricles back into the veins) very high

venous waves periodically appear, the existence of a complete heart block may be assumed

Not rarely *isolated auricular sounds* can be heard in diastole At times they are quite loud and are audible to the left of the lower end of the sternum or at the cardiac apex Sometimes they are confused with the short rumbling diastolic murmur of mitral stenosis But with careful auscultation one hears that the auricular sounds (in contradistinction to a mitral murmur) appear at times earlier, at times later in diastole, and occasionally are absent

Among the most definite signs of heart block is the occurrence of so called cannon sounds If in heart block one listens at the cardiac apex marked accentuation of the first sound is heard in a majority of cases once, twice, or several times a minute, at times it is loud and thumping as in mitral stenosis, so that the designation cannon sound is understandable Investigations in which the heart sounds are recorded at the same time as the electrocardiogram show that these cannon sounds are not heard when the auricles and ventricles contract simultaneously, but only when the auricles precede the ventricles by a few hundredths of a second, this is explained by the dynamic status of the ventricular filling (Selenin and Fogelson) Since such an alteration of the degree of loudness of the first sound does not otherwise appear with *rhythmic* cardiac action, this sign makes the diagnosis possible

If complete heart block is diagnosed, it must be determined whether it is caused by an organic (permanent) interruption or whether merely an increased vagal tone with a slight damage of the A–V system (digitalis) is producing the block which is then capable of retrogression This is accomplished by having the patient perform some exercise (bending of the knees) or, if he cannot work, to inhale amyl nitrite (five drops placed upon a cloth and inhaled for twenty seconds) Through these measures the vagal tone decreases, and the sympathetic tone increases, thereby conduction is transiently improved and the block diminishes unless it is caused by a complete destruction of the A–V system, in which case it remains persistently unaltered

Fig 165a shows complete heart block with a ventricular rate of 84 The block developed during digitalis therapy After 5 drops of amyl nitrite the complete block vanished and, although a prolonged conduction time remained (Fig 165b), all stimuli reached the ventricle A few minutes later the heart block reappeared and vanished finally only after the discontinuance of digitalis therapy The tracing was obtained from a case of severe coronary sclerosis

The automatic beats in heart block (Fig 165a) are identical with the beats conducted after amyl nitrite, this favours the assumption that the automatic beats originated above the site of division of the bundle

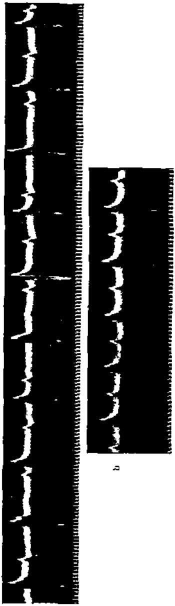

Fig 165a and b In (a) a complete auriculo ventricular block exists which after the inhalation of amyl nitrite passes over (b) into full rhythm with a prolonged conduction time

Cases of partial heart block react very differently to physical exertion The block may become increased or diminished The sympathetic tone increases through exercise and thus the conduction improves But simultaneously the auricular rate is increased, thus the conduction pathway is subjected to greater demands and auriculo ventricular conduction impaired Indeed, according to the factor which predominates, the existing block will be augmented or diminished by work An example of the increase of block by effort is furnished by Fig 159

The assumption of Mobitz that Wenckebach's periods occur only in the A–V node was refuted by the experiments of Scherf and Shookhoff, who succeeded in animal experiments on the heart *in situ*, in injuring the bundle branch at an area remote from the A–V node, and in producing periodicity Subsequently Schellong produced Wenckebach's periods in muscle strips by producing digitalis intoxication

There is one form of partial heart block, *i e*, of periodic dropped beats, which is never the result of increased vagal tone, of a general intoxication or of digitalization, but always due to a circumscribed anatomic cardiac disease It is differentiated from the Wenckebach period by the fact that the conduction time always remains equally long, that is, no increase of conduction time occurs

after a long series of conducted impulses in other words no change takes place Moreover at times several auricular beats may be blocked in succession this never occurs in a Wenckebach period where only one stimulus is blocked It is certain that the intensity and extent of the pathological process in the specific tissue and not the site of the morbid process decides which form of periodic dropped beats occurs

This form of periodic dropped beats which has just been described has been called by Mobitz Type II of periodic dropped beats in order to differentiate it from the Wenckebach period or Type I

Clinical and experimental observations support the suggestion that Type I periodic dropped beats appears in extensive injury of the conduction system while Type II is found only in circumscribed injuries In intoxications for example with digitalis or fatigue of the conduction pathways (*tachycardias*) *only Type I* (*Wenckebach's period*) is found

In Fig 166 at the beginning of Lead I only two of three auricular stimuli are conducted to the ventricle (3 2 block) In contrast to a Wenckebach period with 3 2 block (Fig 155) the conduction time remains constant Then a group of four auricular stimuli follows of these only *two are conducted to the ventricle* (4 2 block) Despite the great differences in the duration of the ventricular pauses here also the *P R intervals remain unaltered* The same is evident in other leads

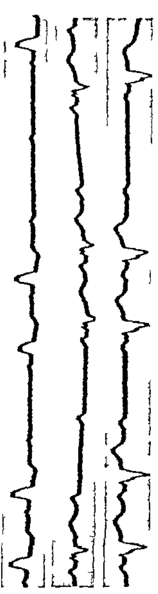

Fig 166 *Type II of periodic dropped beats and disturbance of intraventricular conduction*

Apart from the auriculo ventricular block of Type II a severe disturbance of intraventricular conduction is present in Fig 166 The ventricular complexes show the picture of the common form of bundle branch block

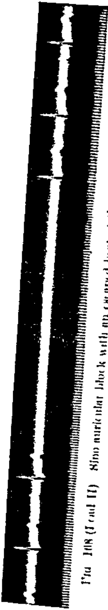

FIG. 168 (Lead II) Sino-auricular block with an escaped beat at the end of the cardiac standstill

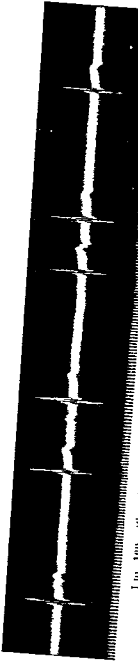

FIG. 169. Sinus bradycardia from sinus block (2 : 1) with the intrusion of escaped beats

FIG. 170 Extreme sinus bradycardia through 2 : 1 sinus block with escaped beats The conducted sinus beats are entirely aberrant owing to abnormal spread within the ventricle

In Fig 169 a very marked but regular sinus bradycardia of 27 beats per minute (length of cycle 2 20 seconds) is present This bradycardia is due to the fact that every other sinus stimulus is not conducted (or not formed ?) In the long pauses an A-V nodal

2 One can also observe a slow automatism in a highly placed block when the pathological process which leads to block also injures the deeper automatic centres Under these circumstances they cannot become active with their customary rate and therefore work very slowly

But it is interesting that in many instances patients who show a ventricular automatism of 30 or less also have strikingly few complaints as long as they do not exert themselves unduly A wonderful compensating mechanism in the circulatory dynamics prevents disturbances It was emphasized earlier that a bradycardia is almost always found in trained athletes The slower the cardiac rate the larger the stroke volume If the stroke volume with a minute rate of 80 amounts to 60 c c with 40 beats per minute a stroke volume of *120* c c is found For the circulation only the *product of these two factors the minute volume has significance*, it is obvious that this may be just as great with a bradycardia of 40 as with a rate of 80 since diastole is longer and the ventricle fills better when the cardiac rate is slow (Lundsgaard) In other words in bradycardias providing they do not exceed a certain limit all disturbances are also completely compensated and clinical experience shows that this holds for rates down to 20 beats per minute as long as the heart and the vessels are otherwise healthy

In very decided bradycardias the heart is enlarged by virtue of the marked filling One often reads in X ray reports of athletes and cases of heart block Plump universally enlarged heart ' However only a dilatation of dynamic origin is present On auscultation of these patients as a rule a systolic murmur is audible over the aorta probably for the reason that owing to the dilatation of the left ventricle the normal aortic orifice acts as a relative stenosis Frequently the systolic blood pressure is increased in *these cases of heart block and bradycardia*

With greater exertion to be sure the circulation of these cases *of heart block all too easily becomes inadequate In the healthy* person with the heart beating in sinus rhythm the cardiac rate is increased by the performance of work through alteration of the vagal and sympathetic tone so that the minute volume increases somewhat and the musculature receives more blood The situation is different in a deep seated heart block The vagus on the whole *has no influence on the centres located deep in the ventricles* (p 20) and the accelerating action of the sympathetics is likewise much less than on the higher centres accordingly after work the cardiac

The *complaints* of patients with disturbances of conduction are remarkably few In most cases the patient does not become conscious of the abnormal state In prolongation of auriculo ventricular conduction the cardiac rate and rhythm on the whole are not disturbed In cases of Wenckebach periods the pause conditioned by the failure of conduction is not sufficiently long to evoke symptoms The pause is hardly ever perceived In higher grades of block the deeper centres become active during the longer ventricular pauses, and through their own rhythm prevent any unpleasant standstill Reference has been made elsewhere to the fact that in complete failure of auriculo ventricular conduction the centres of the ventricle assume control and the patient on the whole does not notice this severe disturbance *Only* when automatism fails for some reason that will be considered below, does the patient become conscious of the disturbance

The rate of automatism of the ventricles in many cases of heart block is as has been mentioned very high Minute rates exceeding 60 are very common But also rates of 80, 100 and more have been observed (Fig 165) The explanation of this becomes evident by the following consideration the A–V node represents a ' physiological barrier ' for auriculo ventricular conduction, even normally it retards auricular stimuli Thus it is conceivable that in heart block due to an increase of vagal tone intoxications or one of many organic disturbances of conduction the site of block is high in the A–V node If the interruption of conduction occurs quite high in the A–V node, an automatic centre highly situated and just below the site of block becomes active This A–V nodal centre has highly developed automatism As a rule its rate is only 15–20 beats per minute slower than that of the sinus node

If in heart block the ventricle beats slowly, two factors may be responsible 1 The interruption of conduction may be located so low that a very deep automatic centre becomes active In a deeply situated block the ventricular automatism can be as low as 30 beats or less per minute When auriculo ventricular conduction is interrupted by a bilateral bundle branch block the automatism may be even less, in rare cases rates of 16 or even 10 beats per minute have been observed (Fig 164)

In cases of complete auriculo ventricular block caused by a bilateral bundle branch block variform ventricular extrasystoles frequently occur (Wilson) This phenomenon is understandable since the same lesion which causes the block may irritate the specific fibres and thus initiate abnormal stimulus formation

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But it is interesting that in many instances, patients who show a ventricular automatism of 30 or less also have strikingly few complaints as long as they do not exert themselves unduly A wonderful compensating mechanism in the circulatory dynamics prevents disturbances It was emphasized earlier that a bradycardia is *almost always found in trained athletes* The slower the cardiac rate the larger the stroke volume If the stroke volume with a minute rate of 80 amounts to 60 c c, with 40 beats per minute a stroke volume of 120 c c is found For the circulation only the product of these two factors, the minute volume has significance, it is obvious that this may be just as great with a bradycardia of 40 as with a rate of 80, since diastole is longer and the ventricle fills better when the cardiac rate is slow (Lundsgaard) In other words in bradycardias, providing they do not exceed a certain limit, all disturbances are also completely compensated, and clinical experience shows that this holds for rates down to 20 beats per minute as long as the heart and the vessels are otherwise healthy

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rate remains far below the actual need Vertigo dyspnœa and states of anxiety then appear

The patient must adapt his performance to the condition of his circulatory system If this occurs he may remain without symptoms for years Patients with heart block who without any complaints follow laborious occupations (porters butchers) are commonly encountered With the disturbance of conduction unpleasant events occur only when Adams Stokes attacks appear They will be mentioned later (p 300)

The appearance of a disturbance of conduction therefore has great significance because in patients who have not undergone previous digitalization it permits the assumption of a myocardial disease even though all other signs are absent The finding of a disturbance of conduction to be sure at first shows only that a pathological focus exists in the A V system Whether it involves an acute or chronic disease an inflammation a necrosis an old scar or degeneration can be determined only by the clinical examination In an old person a simple prolongation of conduction time may be the first indication of the presence of coronary sclerosis and its appearance in rheumatic disease may indicate the presence of myocarditis Like abnormal T waves in many cases the appearance of disturbances of conduction alone also permits the diagnosis of a myocardial disease

Such alterations are often transient and may be absent during one examination But if under these conditions an electrocardiogram is recorded on alternate days on the average every second case of acute rheumatic fever will exhibit disturbances of conduction The disturbance can also be the sequel to diphtheria or rheumatic myocardial disease cured years before that is it is the result of a disease that had been inactive for years It may lack significance if the common muscle is intact Despite the presence of heart block these patients can pursue a laborious life without symptoms or treatment The freedom from symptoms of most patients having disturbances of conduction is an important reason why this condition is diagnosed so rarely indeed it is usually an accidental finding The *prognosis* is provided by the underlying pathology and not by the discovery of a disturbance of conduction alone

The *treatment* of a disturbance of conduction may be omitted in most cases If it is asymptomatic one should not be alarmed by the conduction disturbance itself and should direct attention only to the underlying pathological process

Disturbances of conduction caused by digitalis subside rapidly

when the drug is omitted If an organic disease of the conduction system exists only rarely can it be influenced by medication No remedy is known which prevents diphtheritic necrosis and none for restraining the progress of coronary sclerosis But an endeavour should be made to improve coronary circulation by the administration of vasodilating remedies (preparations of theophyllin papaverine) Even when a gumma has produced heart block specific treatment rarely yields a result since the retrogression of a gumma also leaves a scar which usually impairs auriculo ventricular conduction If the disturbance is due to an old scar all therapy will be useless from the start

In former years treatment was recommended for *every* case of block and atropine was often given for this purpose In this connection it may be said that the effective dose of atropine also produces untoward actions and the continuous administration of *atropine is impossible*

Thus it happens that only in extreme bradycardias and attacks of Adams Stokes the remedies mentioned later are employed (p 305) one does not turn to the prescription pad at once in every disturbance of conduction

Intra-auricular Disturbances of Conduction

Just as disturbances of intraventricular conduction are recognized by splintering and widening of the QRS complexes intra auricular disturbances of conduction may be assumed when splintering and widening of the P waves are found Owing to the presence of pathological foci in the auricles or ramifications of the sinus node the wave of excitation is compelled to spread *abnormally from its site* of origin in the sinus node The *abnormal P waves may be permanent or merely transient according to the presence of continuous* or transient disturbances of conduction

Abnormal P waves naturally can also result from an abnormal origin of excitation (abnormal site of stimulus production in the auricle *auricular extrasystoles) just as widened ventricular* complexes occur not only in disturbances of intraventricular conduction but also in ventricular automatism or in ventricular extra systoles The finding of P waves of varying forms with normal auricular rhythm and constant P–R intervals is in favour of an intra auricular disturbance of conduction and against a disturbance of stimulus formation (Scherf *and* Shookhoff Rothberger and Scherf)

Apart *from* widening and splintering P waves may also exhibit

an alteration of form in the sense of increased amplitude (more than 1 5 mm in height) and flattening they may become inverted and wider (more than 0 1 second) Just as the QRS-complex develops from the sum of potentials of the two ventricles the normal P waves represent a summation of potentials developing from the excitation of *both* auricles

From a negative P wave one may not assume an origin of excitation in the A V node with a stimulus originating in the head of the sinus node at a normal site inverted P waves may appear as the result of abnormal spread of the excitation Since the sinus node is located in the right auricle this chamber is stimulated (0 01 second) earlier than the left so that a slight difference in time normally exists between the excitation and contraction of the two auricles

Abnormally large and split P waves are a frequent finding in mitral stenosis and in emphysema probably in connection with the dilatation of the left or right auricle (Figs 36 136)

According to Winternitz the P wave in cor pulmonale is heightened only in Leads II and III and is neither widened nor splintered it is low in Lead I In mitral lesions the largest P waves are found in Leads I and II They are usually split and widened The authors can confirm this statement It is not as yet determined whether these changes are caused by hypertrophy alone or also by dilatation Some observations are in favour of the assumption that the abnormal P waves of cor pulmonale are caused by hypertrophy and the abnormal P waves of mitral cases also by dilatation

Completely isolated disturbances of intra auricular conduction are rare more frequently they are found in conjunction with disturbances of atrioventricular or intraventricular conduction Under these circumstances they are significant because they indicate that a pathological focus exists not only in the ventricle but also in the auricle in other words a generalized extensive disease is present

Examples of disturbances of intra auricular conduction are seen in Figs 23 and 95

In very rare cases a complete dissociation is found between the two auricles (Scherf and Siedek) The disturbance can also be produced experimentally In this case one finds two auricular rhythms completely independent of each other (inter auricular block)

Sino auricular Disturbances of Conduction

Sino auricular conduction disturbances are rare chiefly because of the presence of a great many connections between the sinus node and the auricle and owing to the large dimensions of the sinus node

Moreover, every single fibre of the sinus node is capable of forming stimuli, so that with destruction of large parts, sufficient tissue capable of function still remains Most cases of sinus block are produced by digitalis therapy, this is comprehensible, because digitalis acts upon all centres and all paths of stimulus conduction

The picture of a sinus block may also be imitated by a disturbance of stimulus formation, that is because at times a stimulus is not formed This type is rare More commonly sinus block can be traced to a disturbance of conduction with normal stimulus formation

Fundamentally similar disturbances of conduction may be found between the sinus node and the auricle to those that exist between the auricle and ventricle The most common and also the *most easily recognized form is a periodic omission of systole (dropped beat) through a block of Type II One or more systoles (of the*

Fig 167 (Lead I) Sino auricular block

entire heart, auricle *and* ventricle) are dropped since the corresponding number of stimuli is not conducted beyond the sinus node *Thus it results in the appearance of complete cardiac pauses whose duration is double, triple, or some multiple of a cardiac cycle* If these pauses are longer, the deeper A–V centres become active if this does not occur because the centres are damaged Adams Stokes attacks appear In the presence of higher grades of sinus block and as the result of the repeated appearance of A–V beats, at times a remarkable tracing develops

If a complete sinus block exists, that is no normal stimuli pass from the auricle to the ventricle, the A–V node is continually active and A–V rhythm appears (p 310)

Fig 167 shows pauses which are exactly the double of normal periods in an otherwise regular heart During these pauses not only the ventricle fails to contract but the auricle is also inactive

In Fig 168 four beats are dropped soon after the beginning, the pause amounts to five times the normal period (5 08 seconds) quite late, only after the lapse of 4 5 seconds, an automatic beat occurs

Fig 168 (Lead II) Sino auricular block with an escaped beat at the end of the cardiac standstill

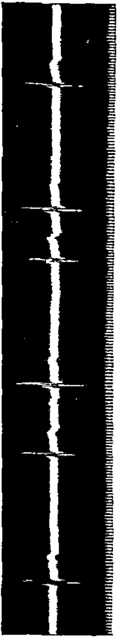

Fig 169 Sinus bradycardia from sinus block (2 1) with the intrusion of escaped beats

Fig 170 Extreme sinus bradycardia through 2 1 sinus block with escaped beats The conducted sinus beats are entirely aberrant owing to abnormal spread within the ventricle

In Fig 169 a very marked but regular sinus bradycardia of 27 beats per minute (length of cycle 2 20 seconds) is present This bradycardia is due to the fact that every other sinus stimulus is not conducted (or not formed ?). In the long pauses an A-V nodal

centre becomes active so that a very peculiar tracing develops In the T wave of the first automatic beat a normal P-wave is seen which is not followed by a ventricular complex since it occurred too early then an automatic beat again appears and soon thereafter a sinus beat with entirely normal conduction Owing to the sinus bradycardia an automatic beat again follows in whose T wave a P wave is concealed which is slowly conducted to the ventricle The next P wave is concealed in the T wave of the following automatic beat and is blocked

In Fig 170 at first glance one might diagnose extrasystoles On closer inspection another disturbance is recognized

A sinus bradycardia of 42 beats per minute is present The first P wave in Fig 170 is seen after the first QRS complex it is hidden in the T wave the next P wave follows immediately behind the third QRS complex and adjoins immediately on an S wave the third P wave follows the fourth QRS complex and has the same position in the cycle as the first P wave the beats of abnormal form which always follow 0 24–0 28 second after a P wave are ventricular complexes of conducted beats (not extrasystoles) Their abnormal form is the result of their prematurity (aberrant conduction in the ventricle) Owing to the marked sinus bradycardia which again is probably the result of a 2 1 sino auricular block escaped beats from the A–V node repeatedly occur

In other words the same disturbance is present as the one reproduced in Fig 169 The peculiar appearance of Fig 170 is merely the result of a different grouping occasioned by other rates of the sinus and of the escape rhythm Likewise the marked bradycardia in Fig 32 is probably the result of a 2 1 or 3 1 sinus block

In sinus block the pauses caused by dropped beats do not always constitute an exact multiple of a normal period since the sino auricular conduction before the dropped beats is prolonged and afterwards may be shortened (as in A V block) Indeed in a Wenckebach s period the pause may be much shorter than double the normal period Exact analysis of such sinus arrhythmias which are independent of respiration is impossible because the activity of the sinus node cannot be directly registered that is changes of the sino auricular conduction are not demonstrable For the same reasons a diagnosis of a simple prolongation of sino auricular conduction—without dropped beats—cannot be made since in such cases the electrocardiogram cannot be differentiated from a normal one

The *treatment* of sinus block is arranged in accordance with the

same viewpoints as the treatment of disturbances of auriculo ventricular conduction In a majority of cases of sinus block of organic origin an affection (sclerosis) of the arteries supplying the sinus node exists

The Adams-Stokes Syndrome

The syndrome which is named after Adams and Stokes[1] is observed when for some reason the circulation stands still and the supply of blood to the brain stops If this standstill lasts only 3–4 seconds usually it is not noticed by the patient only a pause which persists for a longer time causes a sensation of vertigo, after approximately 10 seconds unconsciousness occurs and the patient faints with longer cardiac standstill there is twitching of the arms and legs and finally tonic and clonic convulsions Urine and stool pass involuntarily A standstill persisting for longer than 3–4 minutes is always fatal

This event can be evoked by two mechanisms which are fundamentally different from each other their distinction is important in respect to treatment

According to recent investigations *the first major type* formerly regarded as unusual is much more common than the second and is caused by tachycardia It was stated earlier (p 221) that the higher the rate and the shorter the diastole the smaller the stroke volume becomes If the cardiac rate exceeds a certain limit the time available for ventricular filling becomes too short and the ventricle expels too little blood with too little force so that practically the circulation stands still With a powerful myocardium and with normal vessels otherwise healthy young individuals will tolerate a remarkably high rate extremely well Thus we observed a young man with a tachycardia of 320 beats per minute who was still able to walk slowly to the clinic In older patients with sclerotic vessels in severe heart muscle disease in valvular lesions with predominant stenosis of an ostium and for this reason a small stroke volume from the start even with a lower rate of the heart the circulation may become inadequate for the required perfusion of the central nervous system In a severely damaged heart due to coronary thrombosis even a tachycardia of 180 beats per minute may produce unconsciousness Very often it represents a paroxysmal auricular fibrillation which is not uncommon precisely in old people with sclerosis of the peripheral vessels (p 245)

[1] It was also described by Morgagni

Frequently, despite loss of consciousness, there still exists a slight circulation in these cases since the heart still advances a small amount of blood, so that even a longer duration of the attack is not fatal

The *second main form* of Adams Stokes is encountered in actual cardiac standstill There are various sub groups Most commonly a ventricular standstill appears in disturbances of auriculo ventricular conduction when the conduction to the ventricle fails and the deeper centres do not immediately assume control over the ventricle by their own automatism Normally the deeper centres *are prepared to step in immediately upon failure of conduction* of stimuli to the ventricles If in a healthy dog one sections the bundle of His in a heart exposed by operation, a slight slowing of the heart is seen, but no pause occurs, since an automatic centre immediately below the division becomes active and prevents standstill But if this experiment is performed on another animal with the difference that prior to the severing of the path of auriculo ventricular conduction, a small amount of quinine is injected and thus the automatism of the centres is impaired then they do not function immediately *after the section, rather a certain period elapses until* sufficient metabolites have accumulated in the centres so that they gradually begin to function This pause before the beginning of automatism is called the pre automatic pause The more the specific fibres are damaged, the longer the pause The longer ventricular standstill lasts, the more severe its results

Corresponding to these experimental observations, in most patients in whom a complete heart block appears, the intact deeper centres *always immediately become active and prevent disturbances* But *if the block develops in a case in which the deeper centres are* also injured by the same pathological process which produces the block, for example if arteriosclerosis produces not only the block but also damages the deeper centres, then a pause before the institution of automatism produces an Adams Stokes attack Since complete heart block, after its first appearance, usually does not persist uninterruptedly, as the conduction of stimuli temporarily recovers until it again fails, new attacks recur repeatedly *In the history of patients with heart block the report is frequently heard* that months or years ago frequent attacks of vertigo and fainting occurred One should realize that heart block developed precisely at that time

Fig 171a shows the beginning and Fig 171b the end of an Adams Stokes attack due to ventricular standstill Since it is impossible to employ leads from the extremities during convulsions a chest

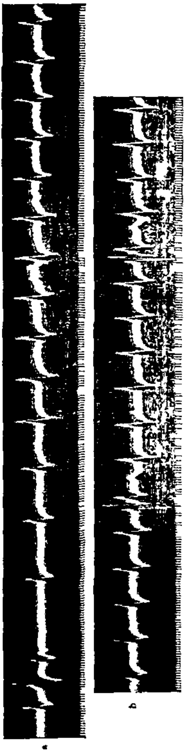

Fig 171a and b (chest lead) Beginning and end of an Adams Stokes attack

lead was used The record was obtained from the right 2nd and 5th intercostal space parasternally In this way remarkably high P waves are obtained which at first glance might be confused with ventricular complexes They are followed by a negative after deflection

At the beginning of the tracing in Fig 171a a ventricular complex may be seen between two auricular waves then all ventricular action fails for one minute and thirty six seconds The auricle beats even more rapidly owing to the insufficient blood supply to the sinus node

In Fig 171b ventricular automatism gradually occurs again The ventricular complexes show an altered form Only after several beats does the automatism reach its full rate

In this case it concerns a rare form of Adams Stokes in a patient already presenting heart block Temporarily the automatism of the ventricular centre fails The block appeared after coronary thrombosis

At all events it must always be assumed that two disturbances are necessary for this form of Adams Stokes to appear (1) the failure of the auriculo ventricular conduction (2) the failure of ventricular automatism

In rare cases a transient cardiac standstill may also develop through a vagal inhibition that is neurogenically There are cases in which such extreme hyperexcitability exists in the receptors in

the carotid sinus that the least pressure at the corresponding area of the neck produces standstill Such patients fall unconscious if *the head is held in a certain position* or if a stiff collar presses on the carotid sinus, cases are also known in which *tumours or* masses of glands press upon the carotid sinus and precipitate most severe attacks (Wenckebach and Winterberg Soma Weiss)

Likewise ' vago vagal " reflexes originating in other places may induce cardiac depression For instance several cases of cardiac standstill due to swallowing have been observed in which a hyper sensitive area in the pharynx was found Cardiac standstill appeared if this was touched with a probe but if the area was anæsthetized with novocaine, the attacks did not occur (Flaum and Klima) In *the discussion of these attacks it must be mentioned that depressant reflexes originating in the carotid sinus may lead to disturbances* of consciousness without cardiac standstill *and without lowering of* the blood pressure (Weiss and Ferris)

The clinical differentiation between the two forms of Adams Stokes (the form with tachycardia and the variety with cardiac standstill with its various sub types) is not always easy The history of the patient is the same in the two forms One might believe that an attack of tachycardia would be assumed when extrasystolic *allorhythmias* are found during the examination of the patient in *the interval between the attacks and that the variety with cardiac* standstill *is present* when outside the attack a disturbance of con duction exists But often this does not hold in practice Thus experience of recent years has indicated that most attacks of circu latory standstill from tachycardia appear precisely in cases with auriculo ventricular conduction disturbances This is conceivable because the same disease which produces the disturbance of con duction also may lead to irritative states in the specific tissues and thus lead to tachycardia *Thus the ventricular fibrillation repro duced in Fig 120 arose in a patient who at the same time had heart* block *Many cases have been reported in which both types of* attack have been observed in the same patient

But the diagnosis is not easy even if one has the opportunity of examining the patient *in* an attack The pulse is absent in both conditions The heart sounds also are inaudible in the tachycardiac form as soon as a certain rate is exceeded On the other hand, during ventricular standstill the regularly working auricle can produce distinct sounds which lead one to *imagine that the ventricle is active* *The examination of the patient in an attack with an* electrocardiogram is desirable But since the attacks appear

suddenly without warning their electrocardiographic registration is not always possible except when attacks are frequent In the latter instance one must apply the electrodes and wait until an attack occurs

From the standpoint of differential diagnosis epilepsy first comes under consideration If in the midst of health the patient suddenly falls down becomes unconscious and convulsions appear this erroneous diagnosis is comprehensible The majority of cases observed by us came by way of the neurological clinic Similar attacks also occur in advanced stenosis of the aortic valve Confusion with simple syncope is common

During the attack of Adams Stokes peculiar disturbances of breathing occur which consist of a severe dyspnœa followed by apnœa This dyspnœa is perhaps the result of an accumulation of abnormal metabolic products in the respiratory centre and the apnœa the result of a preceding hyperventilation in the dyspnœic stage If Adams Stokes attacks become frequent they may be confused with Cheyne-Stokes breathing

Every attack is a serious and dangerous event Everything depends upon whether the blood is supplied to the brain before the centres begin to die Every re awakening from a severe attack is a re awakening from death

Treatment is permissible only when it has been demonstrated whether the attack is produced by tachycardia or by a ventricular standstill Treatment without a definite diagnosis may cause serious injury Both forms require drugs which have exactly opposite actions If cardiac standstill exists stimulating remedies must be given which might be fatal in the tachycardias and in such cases are therefore to be strictly avoided On the other hand the depressant agents effective in the tachycardias may cause serious damage if the attack is caused by cardiac standstill

The therapy of each case is divided into (*a*) the treatment of the attack itself and (*b*) the prevention of new attacks

If the attack is produced by tachycardia at first an attempt is made to abolish the tachycardia by one of the previously mentioned vagal reflexes (carotid sinus bulbar reflex etc) If this is not successful or if a very high ventricular rate exists in auricular flutter or fibrillation where these reflexes are ineffective or only momentarily effective it is best to give strophanthin (¼ mg) intravenously By this means the attack can be stopped (p 268) or at least the ventricular rate reduced But it is clear that immediate aid is not provided with strophanthin since it is effective only after some

minutes moreover it can reach the heart only when the circulation is still sufficient for this purpose As was stated earlier (p 301) this is often the case

It is well to avoid an intravenous injection of quinine in these cases since lowering of blood pressure and decrease of the contractile power of the heart may produce untoward effects But quinine is always preferred when an attempt is made to prevent new attacks The dose under these circumstances would be as discussed in the chapter on tachycardias (p 268)

If an Adams Stokes attack is caused by ventricular standstill one must endeavour during the attack to stimulate ventricular automatism as much as possible A simple and very often effective measure consists in powerful blows in the cardiac region in animal experiments one succeeds easily in awakening automatism through mechanical stimuli

In a case of standstill of the ventricles intramuscular or intravenous injections are useless The intracardiac injection of epinephrine is dangerous because too marked irritation of the centres may lead to the appearance of ventricular tachycardia and even fibrillation A heart in which a block has developed tends to form heterotopic stimuli Ventricular extrasystoles are found with remarkable regularity in these cases Therefore the use of caffeine is recommended for intracardiac injection rather than epinephrine

But if the appearance of new attacks is to be prevented the conduction to the ventricle and ventricular automatism must be improved For this purpose epinephrine preparations are useful One administers ephedrine injections or tablets ($\frac{1}{2}$–1 tablet) three times a day, theophyllin (aminophyllin) is also very effective as a vasodilating agent that increases automatism it is especially effective in those patients whose attacks may be traced to coronary sclerosis

The administration of atropine has proven useful only in those cases whose attacks are ascribed to abnormally strong vagal reflexes and hyperexcitability of the vagus

Repeatedly barium chloride has been recommended for the treatment of cardiac standstill It has been known since the studies of Rothberger and Winterberg that the remedy increases the automatism of the deeper centres to an extraordinary degree If merely a few milligrams of $BaCl_2$ are injected intravenously in a dog ventricular fibrillation appears at once Cohn and Levine recommended very small doses by mouth in order to increase the automatism of the ventricle to the extent that ventricular standstill was

prevented The dose amounts to 40–50 mg $BaCl_2$ which is administered three times a day, that is, 20 drops of a 5 per cent solution three times daily But this therapy has the disadvantage that in different people barium is absorbed from the intestine to a variable extent and at different rates, moreover, the borderline between the effective and toxic dose is very sharp and the margin of safety small Thus at times no action is seen for a long time until suddenly a dangerous heterotopic tachycardia appears Opinions on the utility of barium are divided that of the authors being quite unfavourable The combination of barium with adrenalin-ephedrine preparations is likewise recommended by some authorities but because of the danger of ventricular fibrillation their use should be discouraged

If Adams Stokes attacks appear during the frequent transition from partial to complete block, paradoxically digitalis therapy often aids Conduction is so profoundly impaired by it that a continuous complete block appears Then the automatism of the ventricle becomes continuously active and the attacks usually are no longer feared

One administers small doses [0 2 gm (3 grs) of the standardized powdered leaf] of digitalis daily Since digitalis does not depress the automatism of the ventricular centres but promotes it, this therapy is rational

Even operative treatment (section of one vagus) has been attempted and good results have been reported which perhaps should be ascribed to some other factor, since the improvement occurred only several hours after the operation

One can hardly be too cautious in the appraisal of therapy, since the attacks very frequently cease quite soon even without treatment

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ATRIOVENTRICULAR RHYTHMS AND ATRIOVENTRICULAR ARRHYTHMIAS

The Manifestations of Atrioventricular Rhythm

When the sinus node loses its activity or by virtue of a disturbance of sino auricular conduction the stimuli formed there are not conducted the centres located in the A-V node (secondary centres) assume control over the ventricle

The centres of the A V node possess a well developed automatism Their rate is only slightly less than that of the sinus node so that in the event of an A V rhythm there is only a slight reduction of cardiac rate The patient scarcely ever comes to the physician because of a pronounced bradycardia

The A V node is a poor conductor A stimulus conducted from the auricle to the ventricle is held up for approximately 0 05 second in the A V node so that the auriculo ventricular conduction time normally amounts to the remarkable duration of at least 0 12 second This has a definite practical significance because it permits the auricle to have sufficient time to force blood into the ventricle augment its filling and improve the tension of its musculature

According to the electrocardiographic picture three forms of A V rhythm are differentiated and a different site of origin in the A V node is assumed for each one Against the justification of this localization there are a number of facts based upon experimental observations (Scherf) that it is not proposed to consider in this work The concealed position of the A V node in the heart makes an experimental study of its behaviour in conduction and stimulus formation very difficult At present our knowledge of the physiology of the A V node is very meagre

As long as results of further investigations are not available the customary explanation of the three forms of A V rhythm will despite all objections be retained in order to avoid confusing the beginner

In the most common form of A-V rhythm the auricles and ventricles contract simultaneously the excitation which arises in the middle of the A V node is conducted backward to the auricles and in the normal way to the ventricles Owing to the very poor

conduction in the A–V node, it is very possible for the excitation to require just as much time for the shorter passage to the auricles as for the longer journey to the ventricles As demonstrated in Fig 172b the auricles and ventricles are stimulated simultaneously and the P waves, which appear at the same time as the initial deflections are concealed in them and therefore are invisible

Fig 173 shows in Lead II a tracing of this type A slow ventricular rhythm was seen in all leads P waves were absent They

Fig 172 Schematic drawing of the three forms of atrioventricular rhythm

were also absent when a chest lead was employed (p 150) *in other words* their invisibility does not depend simply upon their small amplitude In animal experiments this form of A V rhythm may be observed directly and the auricles are seen to contract simultaneously with the ventricles

If the stimulus arises high in the A–V node it passes back to the auricles very rapidly and the path to the ventricles requires a somewhat longer time, as is indicated in Fig 172a Owing to this type

Fig 173 Atrioventricular rhythm (middle section of the node)

of spread of the excitation rhythmic cardiac action is seen in which the auricles are stimulated shortly before the ventricles and the P–R interval is very short Since the auricles are excited in a manner directly opposite to the normal the P waves are negative *This form of A–V rhythm is recognized by the fact that in an electrocardiogram negative P waves are followed at a short interval by normal ventricular complexes*

An example of this form of A-V rhythm is reproduced in Fig 174 The cardiac rate is somewhat accelerated (rate 95) Negative P waves are visible before the ventricular complexes which are normal in configuration The conduction time amounts to only 0 08 second

If the stimulus arises in the lower part of the A–V node it reaches the ventricles very rapidly and the conduction back to the auricles through a large part of the A–V node requires a longer time The ventricles as indicated in Fig 172c are activated earlier than the auricles the QRS complexes appear first and the

Fig 174 (Lead II) Atrioventricular rhythm (upper section of the node)

P waves follow Usually a negative P wave is found between the QRS and the T waves

In Fig 175 the cardiac rate is regular No P waves are seen before the ventricular complexes In Lead I slight notching of the T wave is visible and it cannot be definitely determined whether or

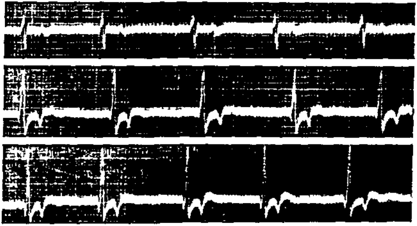


Fig 175 Atrioventricular rhythm (lower section of the node)

not a P wave is present In Leads II and III however deep negative P waves are distinctly recognizable in the S–T interval

Tracings showing the latter two varieties of A–V rhythm (origin of the stimulus in the upper and lowermost sections of the A–V node) are uncommon

An A V rhythm can very easily be produced in the healthy individual under two conditions First it is found commonly during a carotid pressure test (see Fig 1[illegible]1) During vagal inhibition the sinus node loses its activity while the A–V node which is much less

subject to vagal influence, continues to work Second, after the administration of atropine (Wilson), about 30 per cent of normal people transiently show A–V rhythm soon after a subcutaneous or intravenous injection of atropine has taken effect (Eckl) *Atropine produces an "inverse" excitation of the vagus which is evident* for a short time before the paralytic effect becomes apparent Apart from this, atropine does not produce a paralysis of all vagal endings in the heart simultaneously Thus it may happen that for a short time the A–V nodal centres function more rapidly than the centres in the sinus node Since the more rapid rhythm controls the heart, an A–V rhythm is found for the duration of this state In these artificially induced A–V rhythms almost always that variety is present in which the auricles and ventricles are stimulated *simultaneously and the P waves are hidden in the initial* deflections

Parade found a transient A–V rhythm after a cold bath he ascribes this phenomenon to variations in the tonus of the vegetative nerves

An A–V rhythm may be caused in two ways the mode of origin of which is fundamentally different First as already mentioned when no stimuli pass from the sinus node to the auricle that is in an incomplete or complete sinus block In this instance the A–V *rhythm is a sequela of a disease in the region of the sinus node* Second an A–V rhythm is found when an irritant acts upon the A–V node, so that the automatism of the A–V node is higher than that of the sinus node If the cardiac rate is slow (as in Figs 173 and 175), the possibility mentioned first is assumed, but if the rate is high (as in Fig 174), *the second comes under con* sideration

A–V rhythm is frequently referred to as nodal rhythm It is advisable to avoid this designation since sinus rhythm is also a nodal rhythm, *moreover, some time ago fibrillation often was mis* takenly called nodal rhythm (Mackenzie), and this name still appears in older books and adds to confusion

Since in two forms of A–V rhythm the auricles and ventricles contract simultaneously and the auricles thus cannot empty their *contents into the ventricles* the sphincter mechanism at the mouths *of the great veins which normally prevents regurgitation is overcome* and the blood is expelled backward into the large veins *Thus in* A–V rhythm high "superimposed waves" are at times seen in the neck Likewise in A–V rhythm the first heart sound is often strikingly accentuated

No symptoms are produced by an A–V rhythm, the appearance of venous pulsations in the neck is perceived by only a few patients. The normal mechanism produces no enduring circulatory damage, since the cardiac rate, as a rule, is sufficiently slow to permit the ventricles to fill during the long diastoles without the aid of the auricles.

Fig 176 (Lead II) Interference between sinus and atrioventricular rhythm

Interference of Sinus and Atrioventricular Rhythms

If the sinus and A–V centres form stimuli with approximately equal rates and all centres show slight but not completely parallel rhythmic variations, very confusing tracings may appear.

At times a normal P wave is observed at a normal interval *before* the ventricular complex and a sinus beat must be assumed, at times a negative P wave is seen at a normal or shorter interval before or after the initial deflection. "A change in the site of origin of the stimulus" a shifting of the pacemaker appears.

It should be recalled that sinus rhythm may exist even if negative P waves appear at *normal* intervals before the initial deflection, the spread of excitation in the auricles being abnormal, this may result from an origin of the stimulus in the lower sinus node (Rothberger and Scherf).

In Fig 176 varying beats may be observed with normal and negative P waves which are located about 0 15 second before the initial deflection but repeatedly ventricular complexes appear which are neither preceded nor followed by a P wave that is where the P wave is concealed in the initial deflection (see also Fig 179).

Interference Dissociation

In the discussion of ventricular extrasystoles reference was made to the fact that very rarely are they transmitted back to the auricle since they are held up in the A–V node which conducts poorly even in a normal direction. This depression of retrograde conduction also occurs sometimes in A–V rhythm mainly in that form which develops through increased automatism

of an A–V centre, that is in the form which is accompanied by a higher rate At times it is observed in the course of digitalis therapy Usually it is transient

Since the A–V stimuli are not conducted back to the auricle the sinus node works undisturbed

The A–V centre when *functioning more rapidly than the sinus node centre* (otherwise its effect could not even appear) forms independent stimuli ' interfering ' with those of the sinus node Every sinus beat which occurs outside of the refractory phase of an A–V beat is conducted to the ventricle since the auriculo ventricular conduction is then normal The conducted beat breaks into the A V centre, so that its stimulus formation begins anew and a ' coupling of two rhythms ' appears *According to the rate relationships of the*

FIG 177 (chest lead) Interference between sinus and atrioventricular rhythm (interference dissociation)

two rhythms interfering with one another the most diverse pictures develop

Tracings of this type have been known for a long time They were correctly interpreted for the first time by P D White and Wilson, and were named interference dissociation by Mobitz

In *Fig* 177 which was obtained from a strongly digitalized patient who suffered from a mitral lesion an interference dissociation which appeared transiently may be seen The sinus rhythm which was depressed owing to the action upon the vagus of digitalis had a rate of 41 the A–V rhythm a higher rate of 48 Since the A–V beats were not conducted back to the auricle *interference of the two rhythms* occurred At the beginning of the tracing an automatic beat may be seen in whose S–T interval a P wave is concealed immediately after the initial deflection Then an automatic (A–V) beat occurs, the normal P wave which immediately follows on this occasion appears so late that it is conducted to the ventricle although somewhat slowly Then the interference begins again The next normal

Fig 178 Interference dissociation

P wave is concealed in the initial deflection of the A–V beat, the following P waves come increasingly later until one occurs so late that the stimulus is again conducted to the ventricle

A similar tracing is seen in Fig 178, in this case both centres, the sinus node as well as the A–V node, work more rapidly and the difference in rate of the two rhythms is very slight, so that shifting occurs more slowly This tracing also was recorded from a digitalized patient (hypertension and coronary sclerosis), in both cases the disturbance vanished after the discontinuance of digitalis therapy The second and the last but one ventricular complex in Fig 178 are conducted from the auricle, the other complexes originate in the A–V node Clinically one obtains the impression that extrasystoles exist The analysis of the tracings proves however, that the "extrasystoles" are, in reality, conducted beats

Interference dissociation is rare in non digitalized patients In the absence of digitalis it occurs in disturbances of the sinus node when the sinus rhythm is sufficiently slowed as to enable an A–V rhythm to appear The failure of conduction of the stimulus from the A–V node back to the auricle is always a basic condition for the appearance of the arrhythmia At times an interference dissociation is found in a 2 1 sinus block because in this instance the A–V node may work more rapidly than the sinus node

In experimental work an interference dissociation is readily produced by aconitine

If the sinus and A–V rhythms have almost the same rate, but show very slight variations, and if retrograde conduction from the A–V node to the auricle is continually absent, tracings like Fig 179 develop

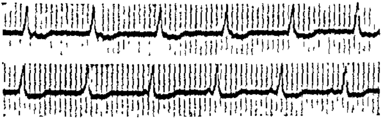
Between Figs 179a and 179b a section of the tracing containing three ventricular complexes has been removed because they dis

played the same form as those at the end of Fig. 179a or the beginning of Fig. 179b. At times the P-waves of the sinus rhythm appear before, at times after the QRS-complexes, and at times are concealed in them. The P-wave is continually positive, that is, the auricle is regularly stimulated by the sinus node. An A–V centre forms stimuli for the ventricle ("competition between two centres") (p. 314).

With marked differences of rate between the sinus and A–V rhythm, tracings appear (Figs. 169 and 170) which it is advisable to consider separately from actual interference dissociation with slow shifting of two rhythms; to be sure, no fundamental difference exists.

In rare cases a centre in the sinus node interferes, not with the A–V centre, but with a centre of stimulus formation lying deeper in the ventricle which works more rapidly than the sinus node. As in Fig. 180, the ventricular complexes of the automatic ventricular

a



b

FIG. 179. *Competition between the sinus and atrioventricular nodes.*

beats are abnormal in shape. In Fig. 180 the sinus node forms 85 stimuli, while the ventricular centre yields 90 stimuli per minute. Here also the ventricles respond to each stimulus from both centres as long as they occur outside the refractory period. The eighth QRS-complex in Fig. 180 is conducted from the auricle, the ninth shows some changes because a part of the ventricle is stimulated by the sinus stimulus and another part by the idioventricular stimulus (summation beat).

Atrioventricular Extrasystoles and Tachycardias

Since, as was stated earlier (p. 165), every specific fibre of the heart is able to form automatic stimuli, and may also be the site of origin of pathologically formed stimuli, extrasystoles and tachycardias may also arise in the A–V node.

Fig. 181 shows extrasystoles occurring between several normal beats; superficial inspection might cause one to diagnose auricular

extrasystoles But closer examination reveals that the auriculo-ventricular conduction of normal beats amounts to 0 15 second, but the P waves appearing prematurely have a P–R interval of only 0 11

FIG 180 Interference between the sinus node and a deep ventricular centre

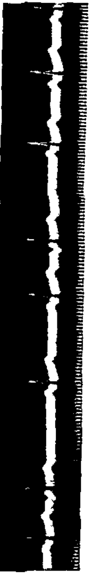


FIG 181 (Lead II) Atrioventricular extrasystoles (originating in the upper part of the A-V node)

second In the discussion of auricular extrasystoles it was stressed that, owing to their prematurity, they are often conducted slowly to the ventricle, so that a prolongation of auriculo-ventricular conduction time appears But, since in Fig 181 premature *negative* auricular waves are followed by ventricular complexes of normal

appearance at a shortened interval, one must assume the presence of auriculo ventricular extrasystoles whose origin is in the upper section of the A–V node

Fig 182 shows a short segment of a record of paroxysmal tachycardia in which likewise inverted P-waves precede the

Fig 182 Atrioventricular tachycardia originating in the upper section of the A–V node

ventricular complexes by very short periods During the sinus rhythm, between the attacks, conduction time was almost twice as long In other words, an A–V tachycardia is present

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PARASYSTOLE

In the discussion of extrasystoles (p 165) it was emphasized that every specific fibre has the ability to form rhythmic stimuli The fibres lying in the sinus node have the most highly developed automatism and the other centres work the more slowly the deeper they are located toward the cardiac apex In the normal heart every stimulus originating in the sinus node on its way breaks into the deeper centres and there destroys all material in the process of stimulus formation so that the automatism of the deeper centres cannot appear

Under abnormal conditions this behaviour can be disturbed A deeper centre may be altered in a way that it no longer responds to a conducted stimulus perhaps, because its irritability is lowered or because the stimulus conducted is weak In this case stimulus formation proceeds undisturbed in this centre which usually lies in the ventricle

Under these circumstances there are two sites of stimulus formation in the heart first, the normal pacemaker in the sinus node, second an automatic centre forming undisturbed stimuli All stimuli formed by these centres which fall outside the refractory period yield a response In a tracing from such a case a regular sinus rhythm and a regular automatic rhythm will be found which work independently of each other Thus it happens that the ventricles may occasionally be stimulated by both (sinus and automatic stimuli) at the same moment because of the continuous interference of both rhythms, the interval between an automatic beat and the preceding sinus beat continuously varies In contrast to interference dissociation where the two rhythms are coupled with each other every time a sinus beat is conducted to the ventricles, here *the two rhythms work entirely undisturbed and completely independent of each other*, as in heart block Kaufmann and Rothberger, to whom we owe the discovery and investigation of these disturbances of normal cardiac rhythm, introduced the names 'pararrhythmia' and "parasystole," since a second independent rhythm prevails in the heart *besides* ('para") the normal rhythm

In Fig 183 superficial consideration suggests the presence of sinus rhythm which is disturbed by isolated extrasystoles But

Fig 181 Parasystole with simple interference of two centres

closer inspection reveals that the time of occurrence of the beats suggestive of extrasystoles varies constantly, those beats at times coming early, occasionally late in diastole, their "coupling," or interval after the preceding normal beat, is at times short, at times long Measurement demonstrates that the interval between the abnormal beats is always a multiple of the interval between two abnormal beats which follow each other directly, that is, *a centre works rhythmically in the ventricle, undisturbed by the sinus rhythm* One finds that some abnormal beats appear more or less simultaneously with sinus beats so that the ventricular complexes show every transition between the two main forms, sometimes a larger part of the ventricle is excited by abnormal automatic stimuli at times by the sinus stimulus so that mixed forms "summation," of the ventricular complexes appear The sinus rhythm has a cycle length of 0 65 second (rate = 92) The automatic ventricular stimuli have a cycle length of 1 22–1 24 seconds (rate = 48 49) The interval in the middle of the tracing that is free from abnormal beats has a length of 4 85 seconds, that is, it corresponds to four times the automatic period If over a period of weeks long sections of tracings are recorded daily, the same situation is always present *Measurement always demonstrates that every automatic stimulus which appears when the ventricle is no longer in a refractory stage after a sinus beat yields a ventricular response*

Indeed according to the relation between the rates of the two rhythms, pictures of the most diverse types of interference appear they can be easily predicted by reconstruction on graph paper

Parasystoles are not rare when one investigates every tracing showing "extrasystoles" which appear in varying phases of diastole in order to see whether or not an automatic rhythm independent of the sinus rhythm, interferes with the latter Actually in parasystole it does not concern extrasystoles because the latter do not develop independently by an automatic stimulus formation, but, as was mentioned earlier (pp 166, 190), are caused by the preceding beat

Therefore the interval between the extrasystole and the preceding precipitating beat in a given case is always the same In parasystole on the other hand the interval between the two types of beats varies

The chief factor in the appearance of parasystole is the undisturbed operation of an automatic centre which is isolated by a protective block (Kaufmann and Rothberger) from other centres of stimulus formation as was stated previously this does not respond to outside stimuli and is not disturbed by them The presence of such a protective block has been experimentally proven (Scherf) Actually it is not a block in the sense understood for many years but an inability of the ventricular centre to respond to outside stimuli

A clear example of a protective block is furnished by Fig 184 Here a complete heart block is present since almost regular auricular waves and regular ventricular complexes appear and the auriculo ventricular intervals vary continually After the second ventricular beat a ventricular extrasystole is seen which *appeared during carotid pressure (the delicate* white line shortly in front of the extrasystole is the signal and indicates approximately the beginning of carotid pressure) As a rule a ventricular extrasystole in heart block disturbs the activity of the automatic ventricular centre since the extrasystolic stimulus breaks into the automatic centre so that it must begin its work anew and the post extrasystolic pause is just as long as an automatic period In Fig 184 this is not the case The automatic centre works undisturbed and its rhythm is not changed by the extrasystole it was protectively blocked

Fig 184 (Lead II) Complete heart block with a ventricular extrasystole which does not disturb the automatic ventricular rhythm it is protectively blocked

As a rule the rate of an automatic centre corresponding to its position in the heart is much slower than that of the sinus node But at times its rate may be higher than that of the sinus rhythm of the affected case Since the more rapid centre always controls the heart a ventricular tachycardia should now appear But it may happen that some of the rapidly

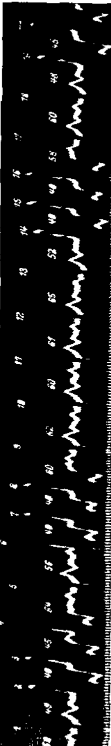

Fig 185 Parasystole with exit block Ie

formed ventricular stimuli elicit no response from the ventricle or are not further conducted (exit block), so that during the ensuing pause sinus stimuli immediately step in Then pictures like Fig 185 may be seen

Here a *sinus rhythm with a rate of* 92–100 is present It is interrupted frequently by groups of 2–3 abnormal ventricular beats which look like extrasystoles Repeatedly mixed (summation) complexes appear The time of occurrence of abnormal beats varies this is against an extrasystolic mechanism and in favour of increased automatism The cycle length of the automatic beats amounts to 0 40–0 45 (mostly 0 40) second and the rate is approximately 150 The three intervals depicted which are filled by sinus beats between the abnormal beats amount to 1 20 (3 × 40) 3 40 (9 × 40) and 1 64 (4 × 41 5) seconds Since in long records this regularity was always found in the same way, the existence of a rhythmically active abnormal ventricular centre must be assumed in this case But the measurement and calculation of the tracing shows that in contrast to Fig 183 not every stimulus formed by the abnormal centre and actually appearing outside the refractory phase of the ventricle elicits a response from the ventricle

A good example of exit block without parasystole is furnished by Fig 186 It was recorded in a case which is repeatedly mentioned in the book of Wenckebach and Winterberg (observation of Scherf Winterberg) It concerns a paroxysmal ventricular tachycardia In Fig 186 a new attack begins after a normal sinus beat, twice the chain of extrasystoles is interrupted The pause thereby developing is exactly twice as long as an extrasystolic period The extrasystoles appear at intervals of 0 45–0 48

second, the two omission periods measure 0 90–0 92 second In other words, there is an exit block and twice the ventricle does not respond to the extra stimulus

This rare form of parasystole which is shown in Fig 185 is called " parasystole with protective and exit block " Simply the relation between the strength of the normally conducted stimulus or of the automatic stimulus on the one hand, and the irritability of the tissue surrounding the centre or of the centre itself on the other is sufficient

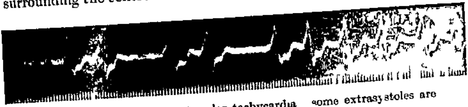

Fig 186 Paroxysmal ventricular tachycardia some extrasystoles are omitted through exit block

to explain the appearance of both forms of parasystole (with and without exit block) It is unnecessary to assume an imaginary block line surrounding the abnormal centre in the ventricle as did Kaufmann and Rothberger

It is interesting that all parasystoles reported up to the present as far as we know, have been found exclusively in patients who have heart disease or in whom the history or the clinical findings permit one to suspect it (Faltitschek and Scherf)

The Bundle of Kent

In lower animals the auricles are connected with the ventricles along the entire A–V junction In mammals this extensive connection is reduced to the small atrioventricular conduction system

The primitive anlage of the heart in mammals represents a " cardiac tube " around which a muscular mantle develops The cardiac tube is the earliest stage in the development of the specific tissue and the superimposed muscle fibres then form the common muscle Here as elsewhere in the heart the developmental stages of phylogenesis are repeated in ontogenesis

In the course of investigations which were designed to determine how the continuity of the embryonic cardiac tube is interrupted during its development, the anatomist Kent (1892) found, in recently littered rats a muscular connection between the right auricle and ventricle Kent was able to demonstrate experimentally the capacity of this bundle for conduction If he sectioned all the connecting

suddenly in the attacks the pulse was irregular Clinical examination revealed an entirely normal cardiac status

The electrocardiogram was abnormal (Fig 187a) There was a

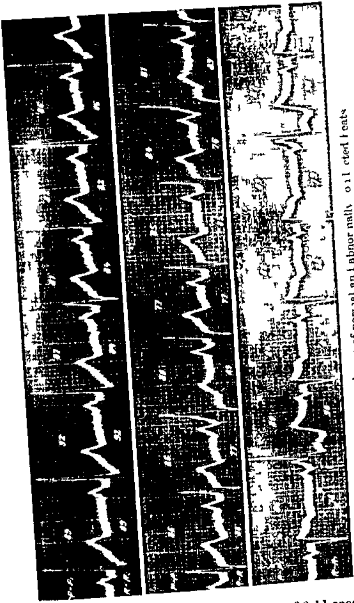

Fig 188 Interchange of normal and abnormally conducted beats

regular cardiac action with a conduction time of 0 11 second and the initial deflections were notched and 0 12 second in width the T waves were also abnormal By means of some knee bending exercises a normal electrocardiogram with a conduction time of 0 17

second could always be produced (Fig 187b) Moreover, the two types frequently alternated with each other

A forty four year old woman reported that for more than twenty two years she had suffered from attacks of palpitation which the electrocardiogram proved to be attacks of paroxysmal auricular tachycardia Likewise in this patient the cardiac examination was normal She also showed at times abnormal and at times normal electrocardiograms

In Fig 188 there is a spontaneous change between the two types Despite a constant sinus rhythm and constant auricular activity, abnormal ventricular complexes repeatedly occur after normal P waves Moreover in these cases the initial deflections are wide and the terminal deflections (especially in Leads II and III) abnormal

On the basis of analysis of these cases it became evident that with constant auricular activity occasionally or for longer periods abnormal ventricular beats appeared *which were always dependent upon the auricles* and which have a shortened conduction time (usually under 0 12 second), it was therefore assumed that in addition to the A V conduction system a second auriculo ventricular path of conduction existed in such cases Where this abnormal pathway of conduction is located is still undecided But since in all known cases of this type after a shortened conduction time *abnormal* ventricular complexes always appear which indicate a disturbance of intra ventricular conduction it was assumed that this second path reaches the ventricle at an abnormal site The duration of conduction in the abnormal path is shorter mainly because the delay of conduction in the A-V node is absent The conducted auricular stimulus can at times employ the normal at times the abnormal path (Fig 188) In one patient the change takes place after a couple of beats in another after months or years Sometimes only abnormal beats are observed Very often one succeeds by means of an exercise test by atropine or amyl nitrite in converting one form into the other Alterations of irritability of the tone of the cardiac nerves result in the auricular stimulus employing the path in which it finds the least resistance

Fig 189a shows alternating normal and abnormal beats About half way in Fig 189a three abnormal beats follow each other In Fig 189b (middle tracing) which was obtained from the same patient a few minutes after an exercise test a bradycardia with marked sinus arrhythmia is recorded Two normal ventricular complexes appear At two places the abnormally conducted beats show a particularly marked widening (immediately before the sinus

beats with normal conduction). It is possible that increasingly abnormal conduction in the ventricle results in the fact that the atrioventricular system is employed by the auricular stimuli.

Fig 189c also shows abnormal ventricular beats following a few hundredths of a second after normal P-waves The *initial* deflections in this case are especially wide and closely resemble the tracings obtained in bundle-branch block

Both cases were normal in regard to the clinical examination of the circulation. In each instance it concerned patients who sought relief for a paroxysmal tachycardia

Since the condition is one of a congenital anomaly (usually such cases are healthy from a cardiac standpoint; the youngest case observed was four-and-a half years old), and since the prognosis is good, up to the present it has not been possible to study the heart of such a case at post-mortem. Only by this means would it be possible to prove the existence of a second auriculo-ventricular connection with certainty. From an embryological point

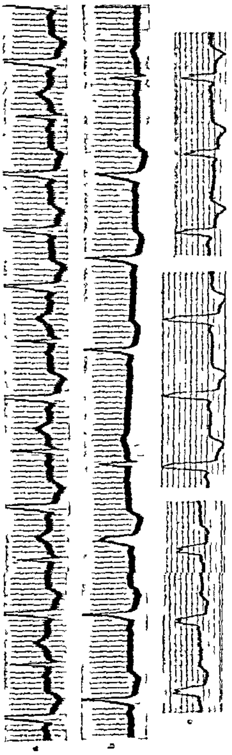

FIG. 189a–c Three instances of abnormal connection between the auricles and ventricles

of view it is indeed possible that at times other remnants of the original extensive connection normally found in the primitive heart may persist which are capable of conduction

It is possible that sometimes retrograde conduction from the ventricles to the auricles is associated with the bundle of Kent this is found occasionally even in complete block of the normal conduction that is in auriculo ventricular block (Averbuch)

Knowledge of this anomaly is important since the physician often overlooks the shortened conduction time and deduces the existence of a myocardial disease from the abnormal ventricular complexes Repeatedly we have seen such cases treated as for myocardial disease and thus unnecessarily disturbed

The ventricular complexes may show quite different pictures

FIG 190 Shortened conduction and abnormal initial deflection in a patient with attacks of paroxysmal tachycardia.

at first glance they may seem abnormal (as in Figs 187–189) or reveal an approximately normal appearance (as in Fig 190) and thus are easily misinterpreted Also the direction of the deflection in Leads I and III can be different However the notch near the base of the ascending limb of the R wave is found very frequently

It is interesting that the majority of cases with abnormal auriculo ventricular connection consult the physician because of attacks of paroxysmal tachycardia Thus 20 of 31 cases of this type complained of sudden palpitation The relationship is not quite clear However it may be recalled that many years ago before cases of this kind were known de Boer elaborated an hypothesis of the development of paroxysmal tachycardia which was based upon the assumption of the existence of the bundle of Kent According to his theory paroxysmal tachycardias develop from a continuous

circulation of a wave of excitation His theory presumes that the excitation in auricular tachycardia uses the following path auricle, bundle of His, ventricle, bundle of Kent, auricle If the excitation proceeds in a reverse direction, i e, down the bundle of Kent and up the bundle of His, then ventricular tachycardia was supposed to occur Some doubt may be expressed regarding this theory (see Fig 207), and the question of the cause of paroxysmal tachycardia (auricular or ventricular tachycardia, fibrillation or flutter) in these cases still remains unanswered

These clinical observations should stimulate further anatomical investigations of the auriculo ventricular conduction in mammals

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THE DIFFERENTIAL DIAGNOSIS OF MYOCARDIAL DAMAGE AND ARRHYTHMIAS

At numerous places in this book it has been pointed out that not rarely the diagnosis of a myocardial affection can be made with certainty *from the electrocardiogram alone* With the assistance of numerous examples it has been demonstrated that the participation of the heart muscle, so common in acute rheumatic fever infectious *diseases* tonsillitis, and coronary diseases, at times can be recognized *only* by means of the electrocardiogram, while the rest of the clinical examination is negative Not rarely the electrocardiogram proves that the *persistent palpitation after tonsillitis or an infectious* disease must be attributed to an accompanying myocarditis that a *vague* pressure in the region of the heart accompanied by marked meteorism is the result of coronary sclerosis, or that a severe coronary involvement exists in a patient affected by syphilis years before although the *present* complaints are trifling

But it was also emphasized that digitalis therapy may alter the electrocardiogram *so profoundly that the picture associated with* severe myocardial injury appears, reference has also been made to the fact that in some normal people who are standing quietly indeed in rare cases *even* when sitting, electrocardiograms are regularly found which must be unequivocally designated as abnormal

It has been repeatedly *emphasized* that *one can* conclude from the electrocardiogram that a myocardial disease is present but no *information as to its nature is obtained* There *is* indeed, *no* electrocardiogram typical of any pathological process The history *and the* physical examination *usually assist in the differential* diagnosis The electrocardiogram supplements the result of clinical examinations but never supplants them

Abnormalities and signs indicating that pathological foci (of some type, recent or old, inflammatory, degenerative, scars, etc) are *definitely* present in the heart *are the following* —

1 Initial deflections which are wider than 0 1 second

2 Marked splintering and slurring of the waves *of the* initial deflection

3 Abnormal S–T segments (observe the pathological displace

ments and when using string galvanometers the disturbing effects of polarization and rule out the action of digitalis)

4 Abnormal T waves in Leads I and II (digitalis !)

5 Variform ventricular extrasystoles

6 All forms of conduction disturbances (when the administration of digitalis can be excluded)

7 Unequivocally abnormal result of the exercise test

As frequent and important signs of a myocardial disease but not absolutely decisive when isolated and susceptible to evaluation only with other clinical evidence, may also be mentioned —

1 Flutter and fibrillation

2 Paroxysmal tachycardias

3 Multiple extrasystoles parasystole

4 A deep Q wave in the third lead

All of the four electrocardiographic alterations just enumerated may also appear in patients who either over a long period or continually do not exhibit any obvious evidence of a cardiac affection But such cases arouse suspicion and always demand a very careful examination and observation

A normal electrocardiogram never proves that the heart is healthy

The *prognosis* of a cardiac malady should never be determined from the electrocardiogram alone

In the following discussion information made available by the preceding sections is amplified with the aid *of additional illustrations*

Fig 191a shows the electrocardiogram of a twenty two year-old male who as a child had suffered from rheumatic fever and chorea Since that time an insufficiency of the aortic valve with a heart of normal size had existed In 1933 examination furnished the electrocardiogram of Fig 191a The initial deflections are 0 08 second in width the T waves in Lead II are low, but there are no *indisputable* signs of myocardial disease The splintering of the initial deflection near the base line in Lead I may also appear normally

Without having been ill in the interim and still free from symptoms the patient returned once again for examination after a lapse of two years

The electrocardiogram recorded at that time is shown in Fig 191b The low splintered P waves have remained unchanged In Lead I a deep and wide S wave follows after a thin R wave the same is seen in Lead II In Lead III a wide double pointed R wave appears after a short Q wave The width of the initial deflection has

increased to 0 13 second The terminal deflections are normal The negative T wave in Lead III also occurs normally

Had a deeper Q wave been found in Lead III, the electrocardiogram would display the complete picture of right bundle branch block described on p 58 But this diagnosis may be made in this instance without a *deep* Q wave in Lead III There are tracings which reveal all the signs of this type of block of the right bundle described by Wilson, although a *deep* Q wave in Lead III is absent Since exceptions to the classical electrocardiogram are found so frequently in normal subjects in Lead III one might assume that a deviation from the customary picture could also be found in pathological electrocardiograms

The question whether a bundle branch block exists in such cases

a b

Fig 191a and b In Fig 191a the electrocardiogram is almost normal In Fig 191b there is an atypical bundle branch block

has a certain practical significance If a block of only the main stem is actually present, one might hope that merely a circumscribed injury has occurred, *one* small focus would not have great significance if the remainder of the myocardium is intact But if the diagnosis of bundle branch block is rejected, the existence of numerous foci somewhere in the ventricle must be considered, that is a diffuse (and prognostically unfavourable) myocardial affection

In Fig 192 the electrocardiograms of six patients are assembled (the three leads are shown beneath each other) and all reveal signs of myocardial disease

At first glance Case 1 seems to show the typical and common picture of a block of the right bundle branch of the Wilson type Nevertheless the presence of Q waves in *all* leads and the rather equal distribution of the widening of the initial deflection in Lead II

suggest the possibility of some other disturbance of intraventricular conduction The terminal deflections are normal in form At all events an intraventricular disturbance of conduction is present

In the second case the initial deflection is 0 10 second in width Splintering near the base line is evident in all leads the Q wave in Lead III is abnormally deep The terminal deflection in Lead I shows a slightly depressed S-T, in Lead II there is a depressed S T as well as a deep inverted T wave The diagnosis of myocardial damage seems indisputable

The electrocardiogram of the third case was obtained from a sixty three year old male who twice had survived an apoplexy with transient hemiplegia In Lead I the electrocardiogram is normal In Lead II the T wave is abnormally small and follows a

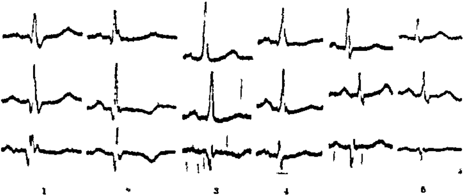


Fig. 107 Electrocardiograms of six cases with signs of a myocardial disease

slightly depressed S T segment The deep wave in Lead III is probably a Q but this diagnosis cannot be made with certainty since a Q wave by definition must always be followed by a distinct R wave But here there is merely a suggestion of an R wave For these reasons one may also suggest that the myocardium is participating in some disease (probably an arteriosclerosis)

The fourth electrocardiogram was recorded in a forty-six year-old patient with malignant nephrosclerosis, gallop rhythm as well as Cheyne-Stokes breathing were present the non protein nitrogen was increased, a short time after the electrocardiogram was taken the patient succumbed to his malady The electrocardiogram does not correspond to the usual picture which is obtained from cases of severe long standing hypertension The left axis deviation with oppositely-directed terminal deflections is lacking Only in Leads I

and II is the plump initial deflection seen (but not in Lead III) the T wave of Lead I is absent, and the low T wave of Lead II indicates unequivocally a myocardial affection Likewise the clinical picture and the subsequent course of the illness justified the assumption of a myocardial disease (presumably coronary sclerosis) which complicated the massive hypertrophy of the left heart

The fifth series is from a sixty three year old patient whose heart was perfectly normal in size and shape, the sounds were pure But upon walking he always suffered from typical radiating pains which immediately vanished upon his taking nitroglycerine Here also it is difficult to say whether or not a deep Q wave is present in Lead III or whether it actually is an S wave In Lead II an unequivocal S wave is present, but this does not prove much since a Q wave may still be present in Lead III Striking and certainly abnormal however, is the low T wave in Lead I

In the sixth series another electrocardiogram is reproduced which resembles a block of the right bundle branch of the Wilson type The findings which suggest it are the plump S wave in Leads I and II, the deep wave generally considered as a Q wave in Lead III (which often but not always is preceded by a small notch) the positive T waves in Leads I and II and the negative T wave in Lead III But, on the other hand it must be stressed that the initial deflections are only 0 10 second in width and the plumpness and width of the S waves in Leads I and II as well as the R wave in Lead III are only slightly evident Such transitional forms are not rare and are interesting If the syndrome described by Wilson actually is a block of the right bundle branch these transitional forms are difficult to understand At all events a disturbance of intraventricular conduction is present in these cases

Sections of tracings of four cases are shown in Fig 193

The electrocardiogram of Case 1 is certainly abnormal The initial deflections are plump, but scarcely 0 10 second in width The S–T segments are depressed below the isoelectric line in all three leads and the T waves are very low In other words, the record shows a kind of ventricular complex that is seen in myocardial damage However a glance at the P waves and the auriculo ventricular conduction time reveals that the P waves precede the initial deflections only by a few hundredths of a second It concerns (owing to the presence of an abnormal P–R interval combined with abnormal ventricular complexes) a case of abnormal auriculo ventricular conduction along the bundle of Kent, that is, a harmless anomaly For this reason it is not correct in this case to assume

myocardial damage on the basis of abnormal ventricular complexes The patient suffered from attacks of paroxysmal tachycardia which are typical of this disturbance

The electrocardiogram of the second case in Fig 193 was recorded in a patient with severe coronary sclerosis and anginal symptoms Two months after the registration of the electrocardiogram shown in Fig 193 the patient died suddenly Plump initial deflections widened to 0 11 second deep Q waves in Lead III and deeply depressed S–T segments with very low T waves in Leads I and II may be seen The deep Q wave and the negative T in Lead III are not enough to make a diagnosis of a posterior wall infarction with certainty Negative T waves in Lead II may occur in healthy individuals The widening of the initial deflection the abnormal

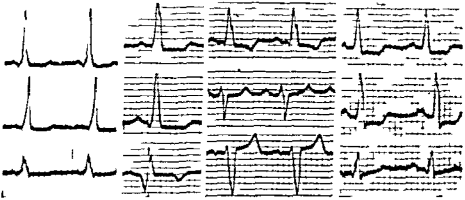


FIG 193 Abnormal electrocardiograms of four patients

S–T segments and the deep Q wave in Lead III are rather in favour of an extensive injury of the heart

In the third case a levocardiogram exists The initial deflections are 0 10 second in width and splintered The terminal deflections are directed oppositely to the initial deflections

One can assign electrocardiograms of this variety to the common form of bundle branch block But it should never be forgotten that another disturbance of intraventricular conduction without a block of the main stem may very easily produce the same kind of tracing A levocardiogram with widening of the initial deflections and abnormal oppositely directed terminal deflections is not rare in myocardial damage In this case a hypertension of 250/140 was present with a very large left heart gallop rhythm pulsus alternans and stasis

If it is asserted that the electrocardiogram of the third case of

Fig 193 shows the picture of the common form of bundle branch block It should not be forgotten first that we do not know *which* bundle branch is blocked and, second that it cannot be unequivocally demonstrated whether or not a main stem of a bundle branch is blocked at all Nevertheless a disturbance of intraventricular conduction is present

In the fourth electrocardiogram high R waves 0 09 second wide may be seen in each lead The S T segments are displaced below the isoelectric line in all leads and the T waves are almost completely absent

The diagnosis of diffuse severe myocardial damage was confirmed by necropsy where an extensive myomalacia with coronary sclerosis was found With more circumscribed pathological processes (myomalacia inflammatory foci) the electrocardiographic alterations are

a b

Fig 194a and b Fig 194a shows the electrocardiogram at rest Fig 194b the electrocardiogram after exercise in a case of coronary sclerosis

usually oppositely directed in Leads I and III and not similarly deflected as in this case

Fig 194 is that of a patient with typical attacks of angina pectoris The pain appeared after exertion of a trifling nature and compelled the patient to stand still They were immediately abolished by amyl nitrite

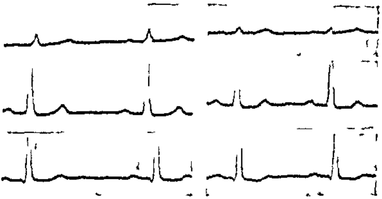
The electrocardiogram in Fig 194a shows a levocardiogram the initial complexes are 0 10 second wide and somewhat plump The T waves are abnormally low in Lead I

Fig 194b shows the electrocardiogram immediately after exercise (climbing stairs 20 metres in height) The T waves in Lead I have become deeply negative in Lead III they are considerably higher In Lead II an auricular extrasystole is visible

Therefore the examination is unequivocally in favour of coronary stenosis Four months later coronary thrombosis occurred necropsy showed an occlusion of the Ramus descendens anterior

Fig 195a was recorded in a forty nine year old patient, who reported that at times, especially after eating, sometimes also on rapid walking he noted a sense of pressure in the "pit of the stomach" The pain did not radiate, was not invariably aggravated by movement and at times persisted for ten minutes The clinical examination yielded normal findings

The electrocardiogram (Fig 195a) shows a conduction time perhaps at the upper limit of normal (most distinct in Lead II), the initial deflection in Lead I is somewhat plump, but otherwise the findings are normal After an exercise test one sees the initial deflection in Lead I somewhat smaller (lower position of the diaphragm), but the depression of S–T in Lead II does not exceed



a b

Fig 195a and b Electrocardiograms before and after work (stomach empty)

the normal physiological range, as often happens in the healthy, the T wave in Lead I is even somewhat higher

On the basis of these results which were obtained by others the patient was treated as having "gastritis" and a corresponding diet and prescription was given He did not improve under the new therapeutic regimen and changed physicians

Since an electrocardiogram during rest and after work again presented the normal appearance (see Figs 195a and b), another electrocardiogram was recorded after eating Distinct alterations became evident (*only* after eating and even before exercise) (Fig 196a) The S–T segment in Lead II is slightly depressed below the isoelectric line and the T wave in Lead II is almost invisible An exercise test conducted immediately afterwards shows a deep depression of the S–T segment in Lead II (Fig 196b), that is, it was distinctly positive

These tracings are instructive for several reasons At first they show how cautious one must be in the appraisal of the result of an exercise test A load upon the heart from climbing stairs did not produce electrocardiographic alterations but after a meal which in itself causes an increased demand upon the heart there appeared severe alterations of the terminal deflections, even at rest but especially after exertion

There are patients who feel anginal pain even after the first mouthfuls of food In other words it does not depend upon the amount of food nor upon a full stomach ' It is probable that vago vagal reflexes which influence the coronary arteries from the digestive tract (Dietrich and Schwiegk) participate in causing the

Fig 196a and b The same patient as Fig 195 before and after exercise (full stomach)

poor blood supply to the heart The overload of the heart by the work of digestion must then be added

Even in their first communications on the exercise test Gold hammer and Scherf made reference to the fact that occasionally climbing stairs alone need not produce electrocardiographic changes whereas the same exertion after a meal may produce distinct altera tions

A slightly accelerated sinus rhythm with a normal conduction time of 0 15 second is present in Fig 197a Lead I shows a deep Q wave followed by a high R, in Leads II and III deep S waves exist The initial deflections are neither splintered nor widened In Lead I the S–T segment departs from the descending limb of the R *very shortly before* the R wave has returned to the base line The T wave in Lead I is positive In Lead II the terminal deflection is

normal in shape in Lead III the S–T segment is depressed slightly under the isoelectric line

The combination of a distinct Q wave, a slightly elevated take-off of the terminal deflection in Lead I and the oppositely directed terminal deflection in Lead III is in favour of an electrocardiogram of the Q_1T_1 type Despite the fact that the alterations are not very pronounced, one may presume the presence of an anterior wall infarction which in that case actually existed

Fig 197a c Fig 197a shows the electrocardiogram after a recent coronary thrombosis 197b the electrocardiogram of a case of pericarditis in 197c a prolonged auriculo ventricular conduction simulated by a disturbance of intra auricular conduction

It should not be forgotten that the presence of a Q wave without very definite alterations of the terminal deflection in the same lead supports the diagnosis of a coronary thrombosis In the absence of clinical signs the diagnosis of coronary thrombosis should not be made solely on the basis of the electrocardiogram, since similar electrocardiograms also may appear in other conditions

In Fig 197b a high take off of the terminal deflection from the descending limb of the R again appears But in this tracing *the alteration is* present in *all* leads and is most marked in Lead II, in coronary thrombosis they are found most distinctly either in Lead I *or* Lead III The electrocardiogram was recorded in a young man suffering from lobar pneumonia with clinical signs of pericarditis

In Fig 197c a prolongation of auriculo ventricular conduction time to 0.22 of a second is noted But more careful inspection reveals that the P waves are so wide (0.11 second) that they utilize about one half of the conduction time In other words the prolongation of conduction is *simulated* simply by a disturbance of intra auricular conduction The conduction time is measured from the beginning of the P wave, so that it includes the conduction within the auricle

The other waves of the electrocardiogram in Fig 197c are normal

The electrocardiogram of Fig 198 was recorded in a twenty six year old female who suffered from mitral stenosis Marked pulmonary stasis was present and severe attacks of pulmonary œdema

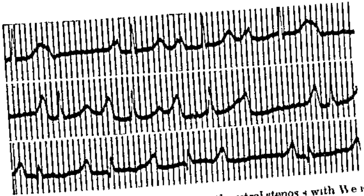


Fig 198 Electrocardiogram in a patient with mitral stenosis with Wenckebach periods

appeared upon any excitement or physical exertion The right heart was not dilated and the left auricle was only slightly enlarged

The electrocardiogram shows Wenckebach periods In all leads may be noted an increasing prolongation of the auriculo ventricular intervals until a ventricular systole is dropped Corresponding to the slight alteration in cardiac size in this case the initial deflections are perfectly normal

The unusually large P waves are also characteristic of mitral stenosis, they attain an impressive height and width especially in Lead II The P–R segment so distinctly depressed below the iso electric line indicates a very large after deflection of the P wave Study of the tracing also reveals that the depression of the S–T segment which is found particularly in Lead II is also caused by the "T of the P"

The disturbance of conduction appeared in this case of mitral stenosis with marked pulmonary stasis after small doses of digitalis and was desirable since it led to a very useful diminution in the cardiac rate and consequently to a longer diastole

In Fig 190 a disturbance of rhythm is present In analyzing the normal (sinus) beats, it is well to begin after a long ventricular pause (before the third R wave in Lead I)

The P waves are normal in all leads Conduction time which is best measured in Lead II, amounts to 0 22 second The initial deflection in Lead III reveals a deep Q wave It is 0 09 second in width and somewhat plump The S–T segment in Lead I is slightly depressed below the isoelectric line and is followed by a very low T wave

One should recall the important rule of measuring the height of

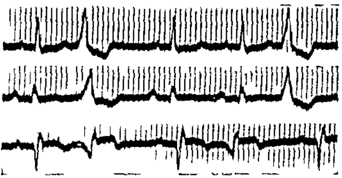


FIG 190 Signs of myocardial disease and ventricular extrasystoles.

the T wave, not from the S–T segment, but rather from the iso electric line (before the P wave of the following beat)

In Lead II the T wave is absent, in Lead III a high take off of the terminal deflection from the descending limb of the R wave passes over into a negative T wave

The curve shows definite evidence of a myocardial affection, the patient reported that four days previously he had suffered from an extremely severe pain which lasted for several hours, it was felt at the lower end of the sternum and radiated upward into the neck The presence of a recent coronary thrombosis which was situated in the descending ramus of the right coronary artery (posterior wall infarction) may be assumed

Moreover, ventricular extrasystoles appeared after every normal beat, at times after every two normal beats, they showed no variation in form According to the rules given in the chapter on the

localization of extrasystoles they probably originated in the right ventricle near the apex

The appearance of ventricular extrasystoles in a case of recent coronary occlusion is always an undesirable complication patients affected in this manner must be watched carefully and treated with quinidine The transition of multiple ventricular extrasystoles into tachycardia is not rare, and the danger of ventricular fibrillation must be considered

Fig 200 is that of a patient who was under treatment for tabes in a neurological clinic One day pain of excruciating severity suddenly developed in the cardiac region and persisted The patient came to the neurological clinic from which he was referred to the

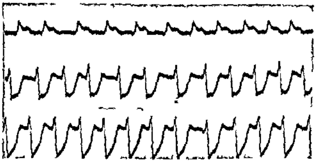


Fig 200 Auricular fibrillation and the electrocardiogram of coronary thrombosis in a patient with luetic coronary stenosis

hospital The history stated that for some time pain had occurred after physical exertion

The electrocardiogram revealed auricular fibrillation with very rapid ventricular rate (170–180) A high take off of the S–T segment from the descending limb of the R wave may be seen in Lead I Alterations are also visible in an unusually clear manner in Leads II and III where there is a low take off of the S–T from the ascending limb of the S waves In conjunction with the history the electro cardiogram is in favour of coronary thrombosis

Without any relief or improvement from therapy, the patient died a few hours later at necropsy it was found that both coronary arteries were quite normal and not occluded But there was a syphilitic aortitis with "such an extreme narrowing of the ostium of the left coronary artery that it was hardly permeable to a sound of hair like diameter" Since the ostium of the right coronary artery was also markedly narrowed one must assume that even at rest only a very limited coronary perfusion was present With the appearance

of paroxysmal auricular fibrillation and the associated rapid rate of the ventricle the disproportion between the oxygen need of the muscle and the oxygen supply was so great that clinically and electro cardiographically the picture of an acute coronary occlusion was simulated

A disturbance of rhythm is also present in Fig 201 The P waves of the normal beats (see Lead II) show varying forms (intra auricular

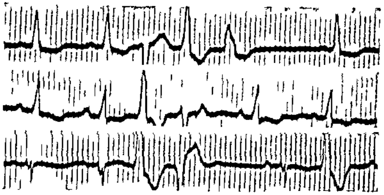


Fig 201 Signs of myocardial disease and ventricular extrasystoles of varying appearance

conduction disturbance) The auriculo ventricular conduction time is normal The initial deflections show a levocardiogram, are 0 09 second in width and appear plump The terminal deflections are directed opposite to the initial deflections, the S–T segments

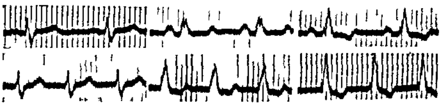


Fig 202 Above a disturbance of intraventricular conduction may be seen the lower tracing shows a paroxysmal (auriculo ventricular?) tachycardia

and T waves accordingly are depressed below the isoelectric line in Leads I and II, while they are above the base line in Lead III

Moreover, ventricular extrasystoles are present and in part are multiple Thus one notes in Lead I three extrasystoles follow soon after the second normal beat The extrasystoles show varying form Since the patient was not receiving digitalis a myocardial injury must be assumed on the basis of the appearance of the extrasystoles

Fig 202 shows in the upper series normal sinus rhythm with

normal conduction time The initial deflections are 0 11 of a second wide, split and plump The terminal deflections are normal in form Accordingly, a disturbance of intraventricular conduction is present In the lower series a regular tachycardia may be seen whose rate to be sure, amounts to only 125 beats per minute P waves are not visible the initial and terminal deflections show the same form in all details as the ventricular complexes during sinus rhythm In other words, a tachycardia is present arising from above the site of the bifurcation of the auriculo ventricular system that is in the A–V node or the bundle of His The attacks lasted from some minutes up to an hour and usually appeared after physical exertion

Fig 203 was obtained from a patient who one month before the electro cardiogram was taken had noted severe pain behind the sternum lasting for six hours In the following few days the typical clinical signs which are usually encountered in a case of coronary thrombosis were noted

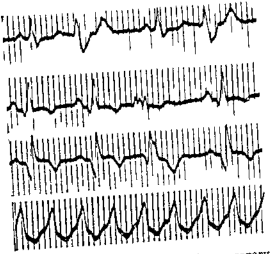


FIG 203 Electrocardiogram of a coronary thrombosis with ventricular extrasystoles The lowermost tracing shows a paroxysmal ventricular tachycardia

In Fig 203 a sinus rhythm is interrupted by numerous extrasystoles The P waves are normal the conduction time amounts to 0 15 second The initial deflections of the sinus beats in Lead III show a very deep Q wave They are 0 10 second wide and plump In Lead I the S T segment is slightly depressed and the T wave is normal In Lead II and especially in Lead III a deeply inverted T wave may be seen Therefore the electrocardiogram is an instance of the T_3 Q_3 type which is ordinarily seen in posterior wall infarction (base of the left ventricle) Moreover, ventricular extrasystoles are present In the lowermost tracing a short section of a tracing from the same patient may be seen in Lead III, it was recorded during an attack of paroxysmal ventricular tachycardia

Auricular fibrillation is present in Fig 204 The ventricular complexes of some stimuli conducted from the auricle to the ventricle

show a levocardiogram and slightly depressed S-T segments in Leads I and II The T waves are very low In addition abnormal ventricular complexes appear (the last three in Lead I, the second in Lead II and the first in Lead III) In all respects these resemble the picture of block of the right bundle branch as shown in Fig 29 and Fig 30 These beats came quite irregularly and accordingly were conducted beats and do not represent extrasystoles

For this reason a partial intraventricular block such as shown in Fig 31 and Fig 32 seems to be present Since one would hardly assume that periodically the conduction *simultaneously* failed in several branches of the conduction system this tracing must be

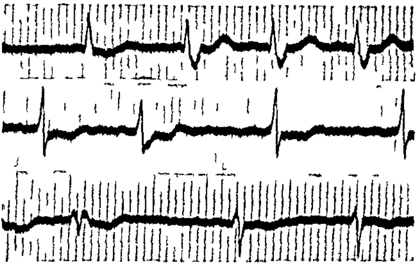


FIG 204 Auricular fibrillation and partial bundle branch block

regarded as proof that the tracings described by Wilson as representing the picture of a block of the right bundle branch actually develop from a disturbance of conduction in *one* conduction path

In Fig 205 after the first and second normal beats two auricular extrasystoles appear and after the third normal beat four extrasystoles are noted The negative P wave of the extrasystole is seen distinctly at the end of the preceding T wave

The first extrasystole of a group is always conducted slowly (prolonged P–R interval) as well as aberrantly (abnormal ventricular complex) to the ventricle This is striking because the first ventricular complex of a group of extrasystoles does not appear more prematurely than the subsequent ones which are conducted normally Thus one sees that the stimulus of the first auricular extra

systole, conducted abnormally in the ventricle usually reaches the ventricle after 0 45 second, but on the contrary, the second, normally conducted auricular extrasystole reaches the ventricle even after 0 40 second

The first auricular extrasystole (the first negative P wave) may well emerge earlier in diastole than the subsequent extrasystoles The ventricle itself or the conduction system below the site of bifurcation of the bundle, however, as measurement reveals, is not reached abnormally early by the first auricular extrasystole

The particularly poor conduction of the first extrasystole is only *one* example of a general law if several conductions follow each other after a long cardiac pause, the second conducted beat finds an especially unfavourable condition of the auriculo ventricular and intraventricular conduction system This behaviour can scarcely be observed with a normal state of the heart But it is distinctly evident when the heart works under adverse conditions of disease or intoxication (digitalis)

The reason for this law seems to be the finding that the first systole after a long cardiac pause is especially powerful and is therefore accompanied by a longer refractory period If the following beat arrives very soon, it finds very unfavourable conditions of conduction All subsequent beats which follow one another after the longer cardiac pause are weaker and have a shorter refractory phase, a shorter recovery period, so that now the conditions for conduction are much better even if many beats (for example, in a tachycardia) appear This law explains why in a paroxysmal tachycardia the first extrasystole of the tachycardia so frequently exhibits a different form from the others and also accounts for the fact that usually in a Wenckebach period the second conduction is much more prolonged in comparison to the first one than the third conduction compared to the second, etc

The electrocardiogram of Fig 206 is certainly abnormal The S–T segments in Leads I and II are

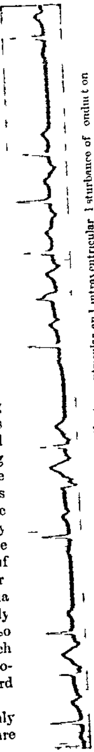


Fig 205 Multiple auricular extrasystoles with atrioventricular and intraventricular disturbance of conduction

depressed below the isoelectric line and the T waves in the same leads are very low. The tracing is that of a fifty three year old woman who two years before had passed the menopause. Severe flushes and palpitation as well as cardiac pain which tormented the patient exceedingly were present. The blood pressure was 16?/80.

The abnormal electrocardiogram in combination with the pain in the cardiac region made the assumption of an organic cardiac disease (coronary sclerosis) seem likely. But recent experiences have indicated that these electrocardiograms are frequently seen in women with ovarian insufficiency and that adequate treatment with follicular hormone not only abolishes the symptoms but also permits the electrocardiogram to return to normal. These electrocardiograms are found in the natural and artificial menopause and in young women with hypogenitalism and disturbances of menstruation.

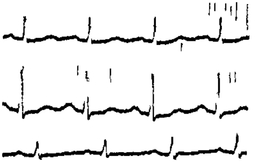


Fig. 206. Abnormal electrocardiogram in the climacterium.

It is probable that the condition is due to a disturbance of perfusion of the myocardium caused by a disturbance in endocrine balance and induced prematurely by the loss of ovarian hormone (hyperfunction of the hypophysis, thyroid and adrenals). One must avoid confusion with the abnormal electrocardiograms of myocarditis, coronary sclerosis, result of digitalis therapy, etc., which are similar in appearance.

Fig. 207 was obtained from a girl ten years old who reported having been well until one year prior to examination she had an attack of whooping cough. Since then attacks of palpitation had occurred; they appeared suddenly and ended just as abruptly after lasting from minutes to hours. Since more recent attacks had been more prolonged and manifestations of congestive failure developed, medical aid was sought.

Fig. 207 shows the three leads during one of the attacks, which, as may readily be perceived from the large positive P waves, were caused by an auricular tachycardia with a rate of 206 beats per minute. The auricular beats were followed by two kinds of ventricular contractions: (1) initial deflections abnormal in shape, wide and splintered, which appeared after a conduction time of

0 08–0 10 second, (2) ventricular complexes which were entirely normal in shape and appeared after a conduction time prolonged to 0 24 second But, as should be stated immediately, this prolongation was the result of digitalis therapy and was never seen when digitalis had not been given. The remaining tracings were not modified by the employment of digitalis, only the T-wave became depressed below the isoelectric line The two types of beats were repeatedly recorded interchangeably in the intervals between attacks, for minutes or hours, during slow sinus rhythm, a normal electrocardiogram or abnormal ventricular beats appeared, the latter after a shortened conduction time All ventricular beats were produced

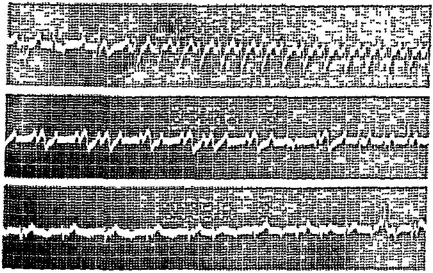


Fig 207 Bundle of Kent (?) with Wenckebach periods

by stimuli conducted from the auricles *In other words, an abnormal auriculo-ventricular connection*, as was described on p 325 (Kent's bundle ?), was present

In Fig 207 normally conducted beats are evident at the beginning of Lead I and at the end of Lead II In Lead III only normally conducted beats are present The disturbance in the second section of Lead III is an artefact.

Conduction between the auricle and the ventricle is disturbed. After a series of normally conducted beats, a 2 1 or 3 2 block recurs repeatedly.

The *tracing* of Fig 207 is of interest for several reasons A paroxysmal tachycardia is present and *during* the tachycardia both

forms of conduction appeared This finding is decisively against de Boer's theory of the origin of paroxysmal tachycardia by the circulation of an excitation wave between the auricle and the ventricle along the bundles of His and of Kent (p 325) Moreover in Fig 207 it may be noted (Lead II) that during the failure of conduction in one path, the other pathway may be employed Of extreme interest is the appearance of a prolongation of the auriculo ventricular conduction time in the second conducted abnormal beat of the two 3 2 block groups in Lead II *In other words a Wenckebach period can also appear in the abnormal path* The second stimulus requires approximately 0 15 second longer for conduction than does the first

We believe that this observation proves that the abnormal beats actually are conducted normal sinus stimuli, this is against numerous other suggestions put forward to explain this disturbance

In the preceding discussion the term "bundle of Kent" has been employed to designate electrocardiograms presenting certain features However it should be emphasized that this is merely an expression of convenience, there is no experimental or clinical evidence that the right lateral bundle is actually or exclusively responsible for the disturbance It is quite possible that some other, as yet unknown, pathway is employed

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